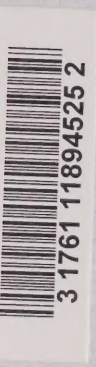

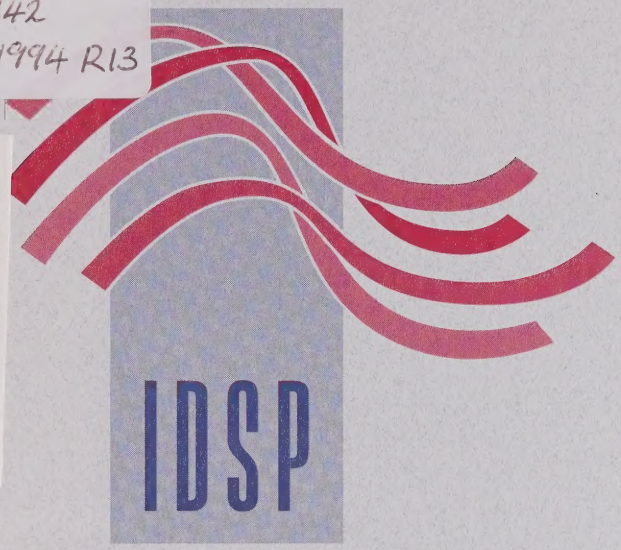

Report to the Workers' Compensation Board on Cardiovascular Disease and Cancer Among Firefighters

September, 1994

Industrial
Disease
Standards
Panel

Report to the Workers' Compensation Board on Cardiovascular Disease and Cancer Among Firefighters

September, 1994

Industrial Disease Standards Panel
IDSP Report No. 13
Toronto, Ontario

Industrial Disease Standards Panel

In 1985 the Ontario legislature established the Industrial Disease Standards Panel (IDSP) to investigate and identify diseases related to work. The Panel is independent of both the Ministry of Labour and the Workers' Compensation Board. At the end of each fiscal year the WCB reimburses the Ministry for the Panel's expenditures.

The Panel's authority flows from section 95 of the Workers' Compensation Act and its functions are set out as follows:

95(8) (a) to investigate possible industrial diseases;

(b) to make findings as to whether a probable connection exists between a disease and an industrial process, trade or occupation in Ontario;

(c) to create, develop and revise criteria for the evaluation of claims respecting industrial diseases; and

(d) to advise on eligibility rules regarding compensation for claims.

Decisions of the Panel are made by its members who represent labour, management, scientific, medical and community interests. Once the Panel makes a finding, the WCB is required to publish the Panel's report in the Ontario Gazette and solicit comments from interested parties. After considering the submissions the WCB Board of Directors decide if the Panel's recommendations are to be implemented, amended or rejected.

To assist with its work, the Panel has a small staff of researchers, analysts and support people. In addition to its own staff, the Panel relies heavily on the advice of outside experts in science, medicine and law, as well as input from parties of interest.

Additional copies of this publication are available by writing:
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Panel Membership

Panel Members	Appointment
Ms. Nicolette Carlan (Chair)	May 16, 1991 to May 15, 1997
Dr. Carol Buck	June 1, 1991 to June 16, 1997
Mr. James Brophy	January 23, 1992 to January 22, 1995
Mr. Robert DeMatteo	April 7, 1993 to April 7, 1996
Mr. William Elliott	November 7, 1991 to November 6, 1994
Ms. Nicole Godbout	December 16, 1992 to December 15, 1995
Mr. John Macnamara	November 7, 1991 to November 6, 1994
Mr. Homer Seguin	May 28, 1992 to May 27, 1995
Dr. Michael Wills	November 7, 1991 to November 6, 1994

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Industrial Disease Standards Panel

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September 1, 1994

Mr. Kenneth Copeland
Vice-Chair of Administration and Chief Executive Officer
Workers' Compensation Board
2 Bloor Street East, 20th Floor
Toronto, Ontario
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Dear Mr. Copeland:

I enclose a copy of the Panel's "Report to the Workers' Compensation Board on Cardiovascular Disease and Cancer Among Firefighters". The Panel plans to issue a report on occupational non-malignant respiratory disease among firefighters in the future.

As you will see, the Panel has recommended that certain additions be made to Schedule 3 and that guidelines be developed. We would be happy to work with WCB staff to develop rebuttal criteria and guidelines, if you agree.

I would be pleased to discuss the Report with you. Please let me know when it would be convenient to do so.

Sincerely,

Nicolette Carlan
Chair

Enclosure

EXECUTIVE SUMMARY

The unions representing firefighters came to the Panel because they believed that there was scientific evidence to establish that firefighting contributes to heart and lung disease. Assuming that the scientific evidence was available the firefighters asked the Panel to recommend that legal presumptions of work-relatedness be applied to workers' compensation claims for heart and lung disease.

As part of its investigation the Panel conducted a study of mortality among Toronto area firefighters between 1950 and 1989. That study revealed statistically significant increases in brain cancer and aortic aneurysm mortality. The rate of cardiovascular mortality was also elevated, although to a lesser degree. The rates of lung cancer were not elevated.

In addition to the mortality study conducted in Ontario the Panel gave careful consideration to similar studies which looked at both the causes of death and the incidence of cancer among firefighters. This evidence was shared with the Unions and the Chiefs of the Fire Departments in the Metropolitan Toronto. After receiving submissions from the workplace parties the Panel developed the following conclusions and recommendations concerning the payment of workers' compensation benefits.

The Panel concluded that :

- **there is a probable connection between cardiovascular disease and working as a firefighter**. Guidelines for the adjudication of such Workers' Compensation claims will be developed.

- **arteriosclerosis which results in aortic aneurysm among firefighters be added to Schedule 3 of the Workers' Compensation** *Act* invoking a rebuttable presumption in favour of such claims.

- **brain cancer and lymphatic and haematopoietic cancers** among firefighters be added to Schedule 3 of the *Act* invoking a rebuttable presumption in favour of such claims.

- **there is a probable connection between colon, bladder and kidney cancer and working as a firefighter**. Guidelines for the adjudication of such Workers' Compensation claims will be developed.

Occupational non-malignant respiratory disease and hearing loss among firefighters will be considered in subsequent Panel Reports.

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CHAPTER 1.
INTRODUCTION

a) The issues before the Panel

The Industrial Disease Standards Panel (IDSP) was asked by representatives of firefighters to determine whether any diseases were attributable to working as a firefighter. This Report focuses specifically upon cardiovascular diseases and cancer among firefighters. Claims for heart attacks experienced by firefighters during the course of their employment are adjudicated under the accident provisions of the *Act* and will not be dealt with in this Report. Occupational non-malignant respiratory disease will be the subject of a subsequent Panel Report.

b) The IDSP mandate and terms of reference

The *Workers' Compensation Act* gives the IDSP authority to investigate possible diseases and when appropriate make findings of "probable connection" between disease and work.

The evidence that the IDSP investigates to find a probable connection is scientific and medical in nature. Specifically, the IDSP considers epidemiological studies, hygiene information about firefighter exposures, toxicological evidence about the identified contaminants and alternative causes of disease.

When evaluating this evidence, the IDSP uses the concepts of Sir Austin Bradford Hill (1965). Bradford Hill argued that to determine causality, consideration should be given to the following factors.

1. strength of association
2. consistency
3. specificity
4. temporality
5. biological gradient
6. biological plausibility
7. coherence
8. experiment
9. analogy

After weighing the evidence, the Panel will decide what, if any, probable connection exists between firefighting and a specific disease. If the results of the investigation do not indicate the existence of a probable connection, the Panel will also report those findings.

When a probable connection is identified, depending on its strength, the Panel may recommend that the WCB take certain steps to ensure compensation payments are made (described below).

c) How these issues arose

In April of 1986, the Ontario Professional Fire Fighters Association (OPPFA) approached the Minister of Labour to request better emergency treatment for firefighters who suffer the effects of inhalation of toxic gases [115]. They were advised to contact the IDSP.

In March of 1988, the Provincial Federation of Ontario Fire Fighters (PFOFF) sent a detailed brief to the IDSP which asserted that firefighters' work subjected them to an increased risk of heart disease [26]. Their brief referred to other jurisdictions which have enacted legislation imposing presumptions that heart and lung disease are causally connected with firefighting.

PFOFF asked the Panel to conclude that a relationship exists between heart and lung disease and working as a firefighter, and to recommend that a legal presumption be enacted in favour of compensation for such claims from firefighters.

The Panel added the issue of cancer among firefighters to its agenda after reviewing the findings of the *IDSP Mortality Study of Fire Fighters in Metropolitan Toronto* (discussed below) and other studies.

d) The organization of this Report

In the following pages, the Panel explores the issues of whether there is a probable connection between cardiovascular disease or cancer and working as a firefighter.

After an outline of the investigations undertaken by the Panel and the stakeholders' views, the legal framework for this work is described. The chemicals which have been identified at fire sites are listed, together with a description of what is known about exposure levels and a summary of the known health effects of each chemical. For cardiovascular diseases and several types of cancer, this Report summarizes the scientific evidence obtained from experts consulted by the Panel and from epidemiological and other data about the rates of disease, dose-response trends and potential causative factors. Ultimately, the Panel explains its policy recommendations which result from the integration of the scientific data and the legal requirements.

e) Investigations by the Panel

After receiving the PFOFF brief, the Panel directed its staff to conduct a review of the world literature on the health effects of firefighting.

That 1988 review identified elevated risks of injury due to falls, burns and accidents, as well as potential acute effects of exposure to smoke and chemicals. The chronic health effects of firefighting were unclear because there were too few studies and the quality of evidence was too limited to answer these questions; however, there was some evidence to "suggest that it is biologically plausible that firefighting could lead to the development of respiratory, cardiovascular and neoplastic disease" [222]. The Panel then placed the issues of cardiovascular and respiratory disease among firefighters on its agenda.

A protocol for a retrospective cohort study of mortality among Toronto area firefighters from 1950 to 1989 was drafted. The protocol was revised [85] in light of comments solicited from four independent expert reviewers [9, 71, 120, 165] and data collection began in January of 1990.

Analysis of those data was carried out and after revisions in light of two additional independent reviewers' comments [66, 57], the final report of findings entitled ***Mortality Study of Fire Fighters in Metropolitan Toronto*** was released in March, 1992 [86].

Because the study identified a statistically significant increase in the rate of brain and nervous system cancers as well as other malignant neoplasms, cancer was added to the other issues on the Panel's agenda.

The firefighters' representatives and Toronto area Fire Chiefs were invited to participate in the process of selecting medical specialists to review the study's findings and the updated literature review. One expert in each medical specialty was chosen by the firefighters, the Fire Chiefs and the Panel to answer specific questions [117, 118].

- about cardiovascular disease:

  Dr. James M. Melius, New York Department of Health [119];
  Dr. Chris D. Morgan, Sunnybrook Hospital [126];
  Dr. John K. Wilson, Canadian Cardiac Health Centres [208].

A list of the questions posed appears in Appendix D.

- about cancer:

  Dr. Norman Boyd, Ontario Cancer Institute [17];
  Dr. Melissa A. McDiarmid, Occupational Safety and Health Administration [111];
  Dr. Ian Quirt, University of Toronto [155].

A list of the questions posed appears in Appendix E.

All of this information was provided to PFOFF, OPFFA, the International Association of Fire Fighters (IAFF), the six Fire Chiefs and two groups of representatives of municipalities for their comments.

PFOFF, IAFF and OPFFA provided written submissions [37, 38, 44, 114] and made oral presentations to the Panel at its meeting of October 30, 1992. Their submissions are summarized below.

The Fire Chiefs sent a representative to that meeting to observe but made no written or oral submissions.

The Municipal WCB Users' Group and the Association of Municipalities of Ontario were invited but both declined to attend or make written submissions.

In response to the firefighters' and some of the medical reviewers' comments, the Panel sought advice from Dr. Bernard C. K. Choi, of the University of Toronto, about how to control for the "healthy worker effect" when interpreting the findings of the IDSP mortality study [24, 25].

Dr. Kristan A. L'Abbé [87, 88], Dr. Paul A. Demers [33] and George A. Tomlinson [193] provided further analyses of the cardiovascular findings. Mr. Tomlinson also conducted additional analyses of the cancer findings and provided advice on the problem of multiple tests of significance [193]. Dr. Melissa M. McDiarmid was asked to elaborate on her previous comments about cancer latency [111].

The Panel sought advice from Dr. Dwayne M. Reed, an Epidemiological Consultant formerly with the Honolulu Heart Program, who has expertise in the etiology of aortic aneurysms [157].

Dr. F. Stern of the US National Institute for Occupational Safety and Health provided additional information about mortality due to aortic aneurysm among New Jersey motor vehicle examiners [187].

Drs. Tee L. Guidotti [58], Paul Targonski [190], John Vena [203] and Kenneth Rosenman [163] were asked to provide further analyses of their data on aortic aneurysms and cancer in other firefighter cohorts.

The Canadian National Centres for Toxicology were asked to comment on the literature about chemical exposures which firefighters may face [20]. In addition, a background paper was prepared for the Panel discussing the toxicology of common chemical constituents of fires and the effect on firefighters' incidence of brain cancer [199].

Finally, Dr. Eric Holowaty and Nelson Chong of the Ontario Cancer Treatment and Research Foundation provided a linkage between data from the Ontario Cancer Registry and the IDSP firefighter study data [140]. George Tomlinson and Dr. Rosa Hong Zhou of the University of Toronto provided analyses of those data [194, 224].

f) Submissions from stakeholders

The PFOFF, the IAFF and the OPFFA provided written submissions [37, 38, 44, 114] and made oral presentations to the Panel at its meeting of October 30, 1992. Excerpts from their unified position follow:

> "Firefighters through the course of their daily duties are exposed to a myriad of substances, which during the course of a fire are exposed to high heat, and mixed together with other substances in unknown quantities....It is our understanding that when a particular substance is tested as a carcinogen it is done so on an individual basis and not mixed with other substances. To do so would be virtually impossible considering all the chemicals known to mankind....how can traditional latency periods be used with any degree of certainty, given the synergistic effect of substances present at a fire?" [114]

> " ... we believe that this study supports the positions of the International Association of Fire Fighters regarding the relationship between fire fighting and disease, based upon prior scientific and medical studies. These positions are as follows:
>
> - First, that fire fighters in the course of their work, are at increased risk of developing acute lung disease and may also be at increased risk of developing chronic lung disease.
>
> - Secondly, that acute cardiovascular disease is exacerbated by fire fighting duties and that fire fighting may increase the incidence of cardiovascular disease in fire fighters.

- Finally, that there is an increased incidence of some specific cancers in fire fighters.

We note, in particular, the evidence in this study supporting an increased risk of chronic bronchitis, emphysema, asthma, cardiovascular disease, and brain and other nervous system tumours, as well as possible increases in cancers at other sites. Besides the elevated SMRs for these conditions, we have noted additional reasons for an etiologic link between fire fighting and these causes of mortality. These include:

- Agreement with prior studies;
- Dose-response and/or latency effects for some conditions; and,
- Biological plausibility provided by laboratory studies.

We have also noted reasons why the link between fire fighting and increased risk for the development of these diseases may have been underestimated. Especially noteworthy was the failure to take into account the healthy worker effect, errors of exposure misclassification, and the limitations of mortality studies in estimating risk of disease." [44]

"It is the position of the International Association of Fire Fighters....that heart and lung disease and cancer be added to Schedule 4 of the *Workers' Compensation Act* for the occupation of fire fighter.[1]" [38]

The IAFF also expressed concern about the risk of noise-induced hearing loss faced by firefighters [39]. They provided copies of studies that suggest that carbon monoxide and solvent exposure can also damage hearing and may potentiate the damaging effect of noise on hearing [4, 105, 45, 46, 168, 223, 134]. Hearing loss among firefighters will be considered by the Panel in a subsequent Report of Findings.

As noted above, neither the Fire Chiefs nor the invited representatives of municipalities made submissions.

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[1] If these diseases were added to Schedule 4 of the *Act*, an irrebuttable presumption of work-relatedness would apply to such claims from firefighters.

CHAPTER 2.
WORKERS' COMPENSATION LAW AND POLICY

a) The legal and administrative framework within which the IDSP operates

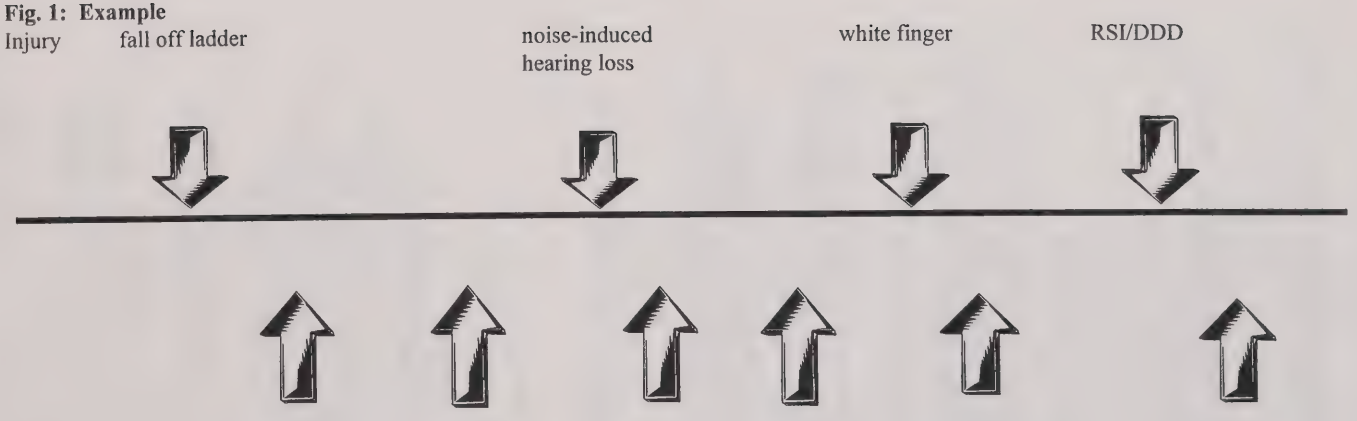
The IDSP's authority to conduct this work is set out in Ontario's *Workers' Compensation Act*. Specifically, the *Act* reads:

> "95. ...
> (8) It shall be the function of the Panel,
>
> (a) to investigate possible industrial diseases;
> (b) to make findings as to whether a probable connection exists between a disease and an industrial process, trade or occupation in Ontario;
> (c) to create, develop and revise criteria for the evaluation of claims respecting industrial diseases; and
> (d) to advise on eligibility rules regarding compensation for claims respecting industrial diseases."

The *Act* also provides the following definition for an industrial disease:

> "1. (1) In this Act,
> ...
>
> `industrial disease' includes,
> (a) a disease resulting from exposure to a substance relating to a particular process, a trade or occupation in an industry,
> (b) a disease peculiar to or characteristic of a particular industrial process, trade or occupation,
> (c) a medical condition that in the opinion of the Board requires a worker to be removed either temporarily or permanently from exposure to a substance because the condition may be a precursor to an industrial disease, or
> (d) any of the diseases mentioned in Schedule 3 or 4."

Fig. 1: Example



CONTINUUM OF INJURIES AND ILLNESS

The term "industrial disease" occurs in the context of the *Act* and refers to an entity defined by the *Act* and not to a medical term. Because the term is defined by the law, the identification of an industrial disease requires the integration of law and science.

The need for and the development of industrial disease policy are distinguishable from other policy development processes at the Workers' Compensation Board. This distinction is visually captured in Figure 1 prepared by the Minister of Labour's Occupational Disease Task Force. Accidents usually are single events with an immediate onset of disability. Diseases normally develop over time and may not be apparent for many years after the initial exposure. For example, asbestos-related illnesses are usually not evident until at least 10 years after the initial exposure and may have an onset as late as 30 years after that first exposure.

Usually, individual workers and employers do not have a sufficiently broad information base to identify patterns of disease. Realistically, the long-term perspective and wide view can only be achieved by scientific research or information gathered by external bodies such as government agencies.

On this point Professor Paul Weiler, one of the primary architects of the IDSP, wrote:

> "Either on its own initiative, or at the request of the Board, the Ministry, or an interested party, the Panel would undertake the review of a disease to which there is, as of yet, no standard, or where the guidelines appear to be outmoded. [At the outset, the Panel would need to canvass a number of internationally recognized industrial diseases for which there is as yet no Ontario standard, although the toxic substance is to be found in Ontario industry: e.g. leukaemia and benzene, angiosarcoma and vinyl chloride, lung cancer and chromium, bladder cancer and the aromatic amines in the petrochemical industry.][2] Probably the Panel would base its work primarily on a review of the world-wide research literature ... " [207]

The IDSP was created in 1985 with the intention of bringing that broad perspective and specialized expertise to the task of identifying industrial diseases.

As set out in section 95 of the *Act*, the Panel is authorized to make findings of **probable connection** between disease and work. These findings are reported to the WCB which has the right to accept or reject

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[2] The text within the brackets is a footnote in the original document.

the findings and to declare the existence of an industrial disease in an appropriate circumstance.

At the time s. 95 was added to the *Workers' Compensation Act*, the Minister of Labour said:

> "I believe there are very few persons who would seriously contest the need for improving our efforts in locating and identifying elements that appear to be causally associated with industrial disease and for developing standards to deal in the fairest manner possible with the compensation claims to which they give rise." [124]

This indicates that the government of the day determined that it would be appropriate for the Panel to make findings of *apparent* causal connection. This suggests that it may be best to understand "probable connection" as meaning something like "apparent causal connection." This should not be confused with the concept of causation, a relationship that is the subject of medical and scientific research.

Once there is a finding of probable connection by the IDSP and a declaration of an industrial disease by the Board, it is then necessary to develop guidelines for the processing of claims. In accordance with its mandate under the law and often at the request of the WCB, the Panel may make recommendations to the Board about when employees with a disease should be compensated.

The *Workers' Compensation Act* has provisions to accommodate the unique issues associated with industrial disease adjudication. Specifically, Schedules 3 and 4 have been added to the legislation to make the workplace parties aware of accepted and declared industrial diseases. By entering a disease into one of these Schedules the work association is declared. Once a disease is assigned to Schedule 3 or 4, a worker need only prove that he or she suffers from the disease and was exposed to the associated industrial process. By demonstrating this, the worker has invoked the presumptions in s. 134. This shifts the onus to the WCB in the case of Schedule 3 diseases to disprove the work association in any specific case. In the case of Schedule 4 diseases there is no ability to disprove an association.

When a disease is not listed in Schedule 3 or 4, in practice the affected worker has the burden of proving the work association. To establish a work relationship the standard applied by the Worker's Compensation Appeals Tribunal, and endorsed by the Minister's Task Force on Occupational Disease, is that work was a "significant contributing factor" to the disease. The worker may be required to present medical evidence, and perhaps epidemiological evidence, about the usual causes of the disease. The worker may also have to

obtain information about the chemicals to which he or she was exposed at work, and investigate whether these chemicals may have "significantly contributed" to the onset of the disease.

It is possible to formulate criteria for determining whether a disease should be added to Schedule 3 or 4 or whether guidelines should be created. However, those criteria have not yet been clearly articulated by the WCB. The best guidance available on this issue are the current entries in each of the Schedules. The Panel has examined the current entries and attempted to identify the criteria used by the Board when entries were made in the past. The Panel has also reviewed internal WCB documents on this point.

When are diseases listed in Schedule 4?

Schedule 4 may be found in the Regulations to the *Workers' Compensation Act*. When a worker suffers from one of the diseases listed in Schedule 4 and can show that he or she was exposed to the associated industrial process, then the following **conclusive** presumption applies:

> "134. ...
>
> (10) If the worker at or before the date of the disablement was employed in any process mentioned in the second column of Schedule 4 and the disease contracted is the disease in the first column of the Schedule set out opposite to the description of the process, the disease shall be conclusively deemed to have been due to the nature of that employment."

Currently, only three diseases are included in Schedule 4: asbestosis, mesothelioma and nasal cancer. The pattern of criteria for inclusion that the Panel has identified by reviewing these entries is as follows:

- a consistent pattern of elevated rates of disease among workers with similar exposures that is substantially greater than the rates in the general population;
- evidence that the rate of disease increases with the extent and/or duration of exposure;
- evidence of known causative substance(s) in the Scheduled work process; and,
- a biological explanation for the development of the disease.

There are no "diseases of ordinary life" included in Schedule 4.

When are diseases listed in Schedule 3?

The legislation setting out Schedule 3 reads as follows:

> "134. ...
>
> (9) If the worker at or before the date of the disablement was employed in any process mentioned in the second column of Schedule 3 and the disease contracted is the disease in the first column of the Schedule set out opposite to the description of the process, the disease shall be deemed to have been due to the nature of that employment unless the contrary is proved."

According to our understanding of the WCB process, when a worker claims to have a disease that is listed in this Schedule, the adjudicator would first determine whether the claimant has the disease and was exposed to the corresponding industrial process. If these conditions are satisfied, the adjudicator would be required to "presume" that the disease is compensable. This does not end the matter, however, since "the disease shall be deemed to have been due to the nature of that employment *unless the contrary is proved*" (emphasis added).

This appears to direct the adjudicator to ask whether there is any other information that demonstrates that the disease was not caused by work. At this stage of adjudication the IDSP believes that WCB staff would be aided by a set of criteria referred to collectively as a rebuttal matrix. That matrix would assist adjudicators in the identification of facts that would lead to the rebuttal of the statutory presumption. If evidence of this sort "proves the contrary", then the disease is no longer presumed to be due to work and compensation may be denied.

Diseases entered in this Schedule appear to have a strong but not exclusive connection to work processes. In determining whether a disease should be included in Schedule 3 the WCB apparently has looked for:

- a consistent pattern of elevated rates of disease among workers with similar exposures;
- evidence that the rate of disease increases with the extent and/or duration of exposure;
- evidence of suspected cause(s) of the disease in the work process; and,
- a reasonable biological explanation for the development of the disease.

For diseases currently listed in Schedule 3 there may be several possible causes for a disease. Currently this Schedule includes diseases which are known to have both employment and non-employment etiologies. This has allowed for the scheduling of "diseases of ordinary life" such as dermatitis.

When a disease is not listed in the Schedules, are there alternative tools available to assist the adjudicators?

Employees receive compensation for a disease if it is established that the disease is "due to" work , even when the disease is not listed in the Schedules.[3] The WCB has issued many "policies" or "guidelines" that assist adjudicators on how to handle claims on an individual basis. These policies are created by the WCB as a result of the investigation of many instances of a specific disease.

In instances where the evidence is equivocal about whether a disease is caused by work, the IDSP has recommended that the Board create specific policies and guidelines concerning the disease. This approach was adopted by the IDSP in its Report on Scleroderma. The lack of certainty about the work association may exist because there is little known about the disease or because there is contradictory evidence about an association. When and if the evidence becomes stronger it may be appropriate for the IDSP to issue revised recommendations to the Board which may endorse entering the disease into a Schedule.

---

3 According to s. 3(1) of the *Workers' Compensation Act*, benefits are payable when workers suffer personal injury by accident arising in and out of the course of their employment.

> "Section 122 of the *Act* provides for the payment of benefits for the consequences of an 'industrial disease' `as if the disease was a personal injury by accident'. Industrial diseases are diseases either listed in schedule 3 or 4 of the *Act* or diseases which have been found to be peculiar to and characteristic of a specific industrial process.
>
> "The legislation also includes the concept of disablement arising out of and in the course of employment within the definition of accident. Previous WCAT decisions, specifically *850* [now reported at (1988), 8 WCATR 73 (Thomas, Acheson, Seguin) at 89] and *559/87* [now reported at (1988), 9 WCATR 103 (Ellis, Lankin, Preston) at 147], have extensively analyzed the interplay between the industrial disease section and the disablement provisions. The end result of that analysis is that compensation is payable if it can be established that the worker's disability arose from a recognized industrial disease as defined by the *Act* or was a disablement caused by an `injuring process', as long as the work process was a significant contributing factor to the development of the disability."[214]

b) Policy and claims experience considerations regarding cardiovascular disease

(i) Ontario

No legal presumption has been applied to cardiovascular disease nor to the occupation of firefighting in Ontario. Claims are adjudicated by the Workers' Compensation Board on a case-by-case basis.

WCB policy on heart conditions states that the Board accepts entitlement if a causal relationship is shown, either as an "accident" or as a "disablement" "arising out of and in the course of employment". That policy includes, as one example of an acceptable causal circumstance, heart conditions which result from "inhalation of smoke and various noxious gases and fumes, e.g., fire fighters" [220]. The full policy is reproduced in Appendix C.

It also provides for conditions arising from unusual physical exertion and/or acute emotional stress as long as there is no significant delay in the onset of symptoms. Claims such as these may be allowed on the basis of aggravation of pre-existing non-compensable conditions, and permanent disability benefits are reduced in proportion to the contribution made by the pre-existing condition.

The Workers' Compensation Appeals Tribunal (WCAT) has allowed claims for fatal heart attacks in firefighters [213], some of which involved pre-existing coronary artery disease or atherosclerosis [216, 215], and in one case the claimant was a smoker [215]. Working as a firefighter was considered a "significant contributing factor", but not the only factor, in the development of the heart condition. In both of these cases, the firefighter was working at the time of the heart attack. Another claim [217] for a myocardial infarction was denied because it did not occur at work and was not considered to be caused by employment exposures.

The WCAT allowed one case involving a fatal pulmonary edema from coronary insufficiency in a firefighter who was a smoker and who had previously suffered a mild, non-compensable cardiac infarction [212]. His death did not occur while working, but while he was undergoing a stress test.

Since 1961, the WCB reports having received a total of 469 claims from firefighters [178]. The types and status of claims for cardiovascular disease are shown in Figure 2.

Fig. 2: Number and status of WCB claims for cardiovascular disease from firefighters since 1961:

DISEASE	CLAIMS FILED	ALLOWED	DENIED	PENDING
Ischemic heart disease	57	24	31	2
Aortic aneurysm	2	1	1	-
Other cardiac disease	8	4	3	1
Cerebrovascular disease	1	1	-	-

During their presentation to the Panel, the representatives of the firefighters stated that firefighters rarely file workers' compensation claims for diseases. It was their perception that the WCB takes considerable time to decide claims for disease, and such claims are likely to be rejected as unrelated to work. Moreover, many firefighters, particularly those in Metropolitan Toronto, are covered by employment insurance plans that provide benefits at least as beneficial as workers' compensation benefits, and there is no requirement that the disease be related to work. These circumstances make it likely that the number of WCB claims filed under-represents the incidence of diseases to which their work may contribute.

(ii) British Columbia

Section 6(3) of the BC *Act* provides:

> "If the worker at or immediately before the date of the disablement was employed in a process or industry mentioned in the second column of Schedule B, and the disease contracted is the disease in the first column of the schedule set opposite to the description of the process, the disease shall be deemed to have been due to the nature of that employment unless the contrary is proved."

In 1954, the BC legislature applied the rebuttable presumption in Schedule B to claims for "injury to the heart" from workers in the firefighting industry. Because the Board began receiving claims from non-firefighters who worked in the firefighting industry, the language in the Schedule was changed in 1980 to cover "[H]eart injury or disease including heart attack, cardiac arrest or arrhythmia, disease of the pericardium, heart muscle or coronary arteries" in claims "[W]here the worker is employed as a firefighter."

A review of reported cases showed that, in practice, the presumption is virtually never rebutted [89].

(iii) Saskatchewan

In response to the Firefighters' Association's position that heart and lung disease in firefighters should be presumed to be work-related, the Saskatchewan WCB commissioned a review of the world literature on heart and lung disease in firefighters, which was completed in 1986 [73]. The author concluded that there was insufficient evidence to show that firefighters' overall risk of heart disease is increased by their occupation and accordingly, no presumption has been enacted. In fact, presumptions are not used in the adjudication of any disease or injury claims in Saskatchewan [167].

(iv) United States

The laws in several US states contain presumptions that heart disease is related to firefighting. Those presumptions are all described as rebuttable; however, because there are widely divergent tests applied to rebut the presumptions, the practical effect varies from irrebuttable to virtually meaningless [91].

c) Policy and claims experience considerations regarding cancer

There is presumptive legislation in some Canadian provinces covering claims for cancer. While some of these presumptions may apply to claims made by firefighters because of their individual circumstances, none of them pertains specifically to firefighters.

(i) Ontario

Schedule 3 of the *Workers' Compensation Act* includes, at entry 4:

"Description of Disease	Process
Epitheliomatous cancer or ulceration of the skin due to tar, pitch, bitumen, mineral oil or paraffin or any compound, product or residue of any of these substances.	Handling or use of tar, pitch, bitumen, mineral oil or paraffin or any compound, product or residue of any of these substances."

There is little doubt that fighting fires involves exposure to compounds, products or residues of tar, pitch, etc. since these are substances which are widely used in construction materials, etc. The WCB has advised the Panel that it interprets "epitheliomatous cancer" of the skin as meaning all skin cancer types including malignant melanoma [42]. This entry provides a rebuttable presumption to firefighters' claims for skin cancer.

The Ontario WCB has no policies about cancer claims specific to firefighters. There are several policies that have identified cancer as an industrial disease, such as lung cancer in gold miners and gastro-intestinal cancer in asbestos workers.

(ii) British Columbia

Section 6(3) of the BC *Act* provides:

> "If the worker at or immediately before the date of the disablement was employed in a process or industry mentioned in the second column of Schedule B, and the disease contracted is the disease in the first column of the schedule set opposite to the description of the process, the disease shall be deemed to have been due to the nature of that employment unless the contrary is proved."

This presumption applies to claims for "[P]rimary cancer of the skin" "[W]here there is prolonged contact with coal tar products, arsenic or cutting oils or prolonged exposure to solar ultra-violet light."

On January 29, 1993, the BC Workers' Compensation Board's Appeal Division granted the claim of a firefighter for malignant melanoma, a cancer of the skin. Although the worker was not able to describe in detail the nature of his exposure over his 22 year career, the Panel considered the available general evidence which indicates that a firefighter is likely to come into repeated intermittent contact with "coal tar products, arsenic or cutting oils". Since, in this case, the contrary was not proved, his disease was presumed to have been work-related [218].

On December 12, 1993, however, that decision was set aside by the Supreme Court of British Columbia on the ground that the Appeal Panel erred by applying too high a standard of proof for rebutting the Schedule B presumption. The case has been returned to the Appeal Division of the WCB for reconsideration [201].

In another case, the BC WCB Appeal Division granted a firefighter's claim for multiple myeloma (cancer of plasma cells in bone marrow[183]). The claim was accepted because the evidence supported a link between the disease and occupational exposure to benzene and because there was no evidence to support any alternate hypothesis [219].

(iii) Manitoba

In 1977, the Manitoba Board passed Regulation #24/77 which stated:

> "4. Where a fire fighter suffers injury to his lungs, brain or kidneys, unless the contrary is shown, the injury shall be presumed to have arisen out of and in the course of his employment as a fire fighter resulting from the inhalation of smoke, gases and fumes or any of them."

That provision was struck down by the Manitoba Court of Appeal in 1988. The Court found that, under the specific terms of the Manitoba *Workers' Compensation Act*, the Board's power to make regulations is limited to administrative and procedural matters and that the Board does not have the power to make regulations which will expand coverage by the *Act*. Consequently, that regulation no longer applies. There have been no attempts by the WCB to introduce guidelines that would apply to claims from firefighters.

(iv) United States

According to our research, twenty-four US states apply presumptions, all rebuttable, to firefighters' claims for cancer but, again, the tests used to rebut those presumptions vary widely.

Alabama, California, Illinois, Minnesota, Nevada, Oklahoma and Rhode Island apply a presumption to claims for "cancer". In Maryland, a presumption is applied to claims for "throat, prostate, rectal or pancreatic cancer, or leukaemia". The states of Louisiana, New Hampshire, North Dakota, Oregon, Pennsylvania, Washington, Missouri, Wisconsin, Virginia, South Carolina, Michigan, Maine, Hawaii, Iowa and Tennessee apply a presumption to claims for lung or respiratory disease [116], which is usually held by the courts to include lung cancer [91].

In 1990, the state of Massachusetts applied a rebuttable presumption in favour of work-relatedness to firefighters' claims for "any condition of cancer affecting the skin or the central nervous, lymphatic, digestive, haematological, urinary, skeletal, oral or prostate systems" [108]. That presumption was applied after researchers at the University of Lowell in co-operation with the Massachusetts Department of Public Health reported findings from their study of the incidence of cancer among firefighters from 1982 to 1986. Those findings were acknowledged to be conservative estimates because about 50% of Cancer Registry records did not indicate any occupation.

The terms of the Massachusetts legislation require active firefighting duty and reflect internationally recognized knowledge about toxicology and latency in the development of cancer. Specifically, it provides that:

1) the firefighter must have undergone a pre-service examination which did not reveal cancer;

2) the firefighter must have served for at least five years before diagnosis, and must have regularly responded to fire calls;

3) the type of cancer must be one which may result from exposure to heat, radiation or a known or suspected carcinogen as determined by the International Agency for Research on Cancer (IARC); and,

4) the presumption ceases to apply five years after the firefighter leaves active fire service.

Despite its efforts, the Panel was unable to identify the reasoning behind the application of legal presumptions in other US and Canadian jurisdictions.

Table 1: FIRES, CALLS, INJURIES AND FATALITIES AMONG METROPOLITAN TORONTO FIREFIGHTERS FROM 1983 TO 1989, BY MUNICIPALITY

	City of Toronto	Scar-borough	East York	North York	City of York	Etobicoke	TOTAL
1983:							
fires	2541	718	250	1173	224	624	5530
other calls	24778	84	2636	12177	24	8	39707
FF injured	138	108	9	117	14	4	390
FF killed	-	-	-	1	-	-	1
1984:							
fires	2428	740	258	1144	255	544	5369
other calls	33850	52	3242	16272	54	12	53482
FF injured	153	48	12	128	19	4	364
FF killed	-	-	-	-	-	-	-
1985:							
fires	2398	924	272	1120	198	545	5457
other calls	30138	10495	2894	14425	33	7	57992
FF injured	172	27	3	135	10	4	351
FF killed	1	-	-	-	-	-	1
1986:							
fires	2247	803	243	1103	256	561	5213
other calls	31197	11323	2997	15190	32	23	60762
FF injured	216	68	4	131	18	4	441
FF killed	-	-	-	-	-	-	-
1987:							
fires	2877	914	244	1108	197	625	5965
other calls	32828	11977	3056	16903	34	15	64813
FF injured	245	90	12	196	7	8	558
FF killed	-	-	-	-	-	-	-
1988:							
fires	2751	895	254	1145	191	523	5759
other calls	35040	13523	3268	19000	46	19	70896
FF injured	258	104	10	132	3	6	513
FF killed	-	-	-	-	-	-	-
1989:							
fires	2634	831	217	1139	251	526	5598
other calls	37706	14940	3693	21115	57	18	77529
FF injured	316	74	21	118	8	24	561
FF killed	-	-	-	-	-	-	-

CHAPTER 3.
THE EVIDENCE

a) About firefighting

Fire protection in Ontario is a municipal responsibility. As of 1986 (when the most recent figures were available to the Panel), 706 of the approximately 800 municipalities in Ontario operated 656 fire departments. Those Departments employed 9,127 full-time and 16,994 part-time (volunteer) firefighters, for a total of 26,121.

Table 1 shows the number of fires, other calls, firefighter injuries and line-of-duty deaths from 1983 to 1989 in each municipality included in the IDSP study. ***It is important to note that firefighters' illnesses and deaths which were caused by diseases are not included in Table 1.*** It is clear, however, that some municipalities responded to many more actual fires than others [135, 136].

There are two main phases of firefighting. The process of extinguishing the main fire is called the "knockdown" phase. The "overhaul" phase involves searching for and extinguishing hidden fires.

Self-contained breathing apparatus (SCBA) were introduced in the last 20 years or so [60]. SCBA includes a pressurized bottle of air carried on the firefighter's back. A hose leading from the air tank feeds clean air into a mask covering the face. Because of its "positive pressure", any leaks flow *out* from the mask, rather than allowing contaminated outside air to enter the mask. When used properly, SCBA provides very effective, but not complete, protection from carbon monoxide and other chemical exposures [101, 94].

Most SCBA provides oxygen for thirty minutes [61]. Since firefighters must allow about ten minutes to get out of a burning building in order to change air tanks, and since the air supply is consumed more quickly during heavy exertion, each tank effectively provides only 15 minutes' breathing protection. The firefighters' representatives explained that there is often an inadequate number of air tanks available to them at fire sites. They said that, in recent years, Toronto fire departments have been equipped with compressors which produce air for refilling used tanks, but that these are rarely if ever available to firefighters who work outside Metropolitan Toronto.

SCBA were as generally under-utilized or used inconsistently in many fire departments until the 1980's [59]. The firefighters' representatives advised the

Panel that in recent years most of them use SCBA during the knockdown phase. Once the main fire has been extinguished, it *appears* that the danger has been reduced and firefighters often remove their SCBA because it is heavy, hot and cumbersome [19]. Its use actually *interferes* with breathing, particularly during strenuous work [102] and even moreso when the tank's air supply has been reduced to 30% of capacity [110]. Regular use of SCBA is particularly difficult under extreme weather conditions [59].

Firefighters who remove their SCBA during overhaul work could suffer the most dangerous exposures [60]. Researchers who have measured carbon monoxide levels in blood (carboxyhemoglobin) report that intermittent use of SCBA offers as little protection as no use at all [94, 95, 60].

It is estimated that 80% of firefighters' injuries are due to smoke inhalation or oxygen deficiency and that over 50% of line-of-duty deaths are due to smoke exposures [60].

Smoke is a suspension of carbon particles in air and in other gases [166]. *All* smoke is hazardous and is potentially lethal at high enough concentrations. The degree of hazard depends on the chemistry and quantity of gases, concentrations reached, size of particulate, solubility of gases and duration of exposure [60].

Particulates become adsorbed (coated) with chemicals in smoke and carry those chemicals deeper into the lungs during active firefighting than they would during normal, less strenuous activities. The heavy exertion demanded by fighting a fire causes more rapid and deeper breathing which increases delivery of toxins to deep within the lungs [60].

Carbon monoxide is a by-product of *all* fire and is one of the most hazardous chemical exposures encountered by firefighters. Since carbon monoxide is odourless, colourless and tasteless, the amount present at a fire site cannot be judged by the firefighter. There is no correlation between the apparent intensity of smoke and the amount of carbon monoxide in the air [60, 94, 95].

Other dangerous products of combustion also continue to be chemically reactive after the main fire has been extinguished and they continue to form additional chemical substances [60, 30]. For example, one of the fuel components of upholstery, wire, pipe coating and wall, floor and furniture coverings is polyvinyl chloride. When these materials are burned, Hydrogen chloride and phosgene are produced as decomposition products [60].

Synthetic materials, such as polyethylene and polyvinyl chloride, have been widely used since the 1950's in furniture and building construction [60].

These substances are often more dangerous when they are smouldering than in high heat. In addition to carbon monoxide [41], synthetic materials cause large numbers of other hazardous chemicals, such as hydrogen cyanide and hydrochloric acid, to be present at fire sites [60, 40, 83, 210]. Moreover, concrete retains heat and gasses, acting like a sponge, then releases toxic fumes as cooling takes place and for long after the fire has been extinguished [60, 40].

- **Chemical hazards of firefighting**

Fire smoke has so far not been well characterized [119]. The chemicals present at fire sites are extremely variable depending upon the type of fire and local physical conditions [60]. As their representatives told the Panel, firefighters can be exposed to a myriad of substances in unknown quantities, which are heated and mixed together [114]. The synergistic effects of these substances are unknown.

In this Report, the Panel has focused on those chemicals which have been identified at fire sites and for which there is evidence of a potential link with cardiovascular disease or cancers. The chemicals discussed below,

- were measured in studies of actual fires as reviewed by McDiarmid and colleagues [112];
- were reported to be present in a subsequent survey of the fire environment by Jankovic et al. [82]; or
- were included in two toxicological reviews prepared for the Panel [20, 199].

The following discussion summarizes the reported levels of these chemicals at fire sites and includes, for comparison purposes, a brief description of their current Ontario exposure limits. (In some cases the exposure limits are too detailed to be included in the text of this report and the reader should consult the appropriate Regulation.) The Ontario Ministry of Labour's bipartite Occupational Exposure Limits Task Force conducted a review of the current exposure limits and has recommended lowering all of the current limits listed below [137].

Most of the limits given below are Short-Term Exposure Values (STEV) or Ceiling Exposure Values (CEV). The STEV is a 15-minute time-weighted average concentration which may not be exceeded at any time during a workday, while a CEV is the maximum airborne concentration of a chemical to which a worker may be exposed at any time during a workday.

It should be noted that an STEV is set on the basis of preventing the acute adverse effects which have been observed in humans or animals after high

short-term exposures. Thus, comparisons between firefighters' exposures and STEVs or CEVs may have little significance in estimating the risk of long-term health effects such as cancers and chronic cardiovascular diseases.

Where an STEV or CEV is not available, a Time-Weighted Average Exposure Value (TWAEV) may be mentioned instead. A TWAEV is the average airborne concentration of a chemical agent to which a worker may be exposed in a workday or work week.

In most cases, a comparison between airborne contaminant measurements at a fire site and a TWAEV is inappropriate because of firefighters' relatively short period of exposure to airborne contaminants at most fires.

Several of the chemicals mentioned below (i.e. acrylonitrile, benzene, asbestos and vinyl chloride) are also regulated as designated substances in Ontario. This means that permissible exposure levels, methods of use and control in the workplace are specifically prescribed by Regulation.

The Panel's work has been aided by critical reviews and evaluations conducted by the International Agency for Research on Cancer. IARC has used its unique international position to develop a system for classification that has been praised for the elegant scientific criteria used for selecting and evaluating published evidence on cancer. IARC is widely recognized as an authoritative source of information on the carcinogenicity of chemicals and complex exposures. For a detailed description of the IARC criteria, please see Appendix A.

IARC's programme, initiated in 1971, relies on international working groups of scientists expert in the particular area under investigation. Information is analyzed from animal studies, other relevant biological data, and case reports and epidemiologic studies in humans. The working group then makes an overall evaluation of the carcinogenicity of the particular agent to humans and the substance is designated as falling within one of four main IARC groupings as listed below.

Group 1 ***The agent is carcinogenic to humans.***
There is **sufficient evidence** of carcinogenicity in humans. A causal relationship has been established between exposure to the agent and human cancer.

Group 2A ***The agent is probably carcinogenic to humans.***
This category is used when there is **limited evidence** of carcinogenicity in humans and **sufficient evidence** in experimental animals.

Group 2B ***The agent is possibly carcinogenic to humans.***
This category is generally used when there is **limited evidence** in humans in the absence of **sufficient evidence** in experimental animals.

Group 3 ***The agent is not classifiable as to its carcinogenicity to humans.***
This category is used for agents that do not fall into any other group.

Group 4 ***The agent is probably not carcinogenic to humans.***
This category is used for agents for which there is evidence suggesting lack of carcinogenicity in humans together with evidence suggesting lack of carcinogenicity in experimental animals.

The Panel wishes to emphasize that the following alphabetical list is far from being an exhaustive list of firefighters' potential exposures.

Acrolein

Acrolein is present in most fires as a combustion product of wood, cotton, carpeting and upholstery [60]. Its vapour may also be found at fire sites where acrolein is stored and used for the manufacturing of products such as metals, plastics, perfumes and methyl chloride refrigerants [20].

In a study of various building fires, 56% of the measurements reported for airborne acrolein were above 3 ppm (ranging from below detection to 98 ppm) [198]. These measurements exceed the STEV of 0.7 ppm (0.3 mg/m3) for airborne acrolein by at least four times [143].

A later study of various types of fires reported lower levels during different phases of firefighting, ranging from not detectable to 3.2 ppm in the knockdown phase and 0.2 ppm in the overhaul phase [82]. The study also showed that significant exposure to acrolein could occur amongst firefighters even with the use of self-contained breathing apparatus since levels as high as 0.9 ppm were measured in air samples collected from inside the masks of these devices worn by firefighters at fire sites.

Acrolein is a severe eye and respiratory tract irritant. It has also been shown to interfere with lung function in animals [20]. The carcinogenicity of acrolein has not been well investigated and, according to IARC, the evidence in both humans and animals is inadequate (Group 3). One of its metabolites, glycidaldehyde, is considered to be carcinogenic [79].

Acrylonitrile

Acrylonitrile is a flammable liquid used in the manufacture of acrylic fibres and various rubber products. At fire sites, firefighters can be exposed to vapours from heated acrylonitrile or from the combustion of products in which acrylonitrile is an ingredient [12].

Acrylonitrile is irritating to the skin, eyes and respiratory tract. It is metabolized to form cyanide which inhibits respiratory enzymes and can cause death [154].

Systemic effects are non-specific but may include the central nervous system (headache and nausea) and hepatic (liver dysfunction), renal, cardiovascular and gastrointestinal (diarrhoea and vomiting) systems [154].

Acrylonitrile has been described as carcinogenic by some researchers [191],

particularly in lung and prostate cancers [176]. IARC classifies acrylonitrile as Group 2A, probably carcinogenic in various cancers such as lung, prostate, stomach, colon, brain, lymphatic and haematopoietic system [79].

Acrylonitrile is a "designated substance" in Ontario. The conditions under which most workers may be exposed to it are set out in Ontario Regulation 733/84 under the *Occupational Health and Safety Act* [145].

Asbestos

Because of its insulating qualities, asbestos has been widely used in building materials for residential, commercial and industrial settings [160]. Therefore, asbestos fibres are likely to be present in a wide variety of fires.

In a 1990 study of 226 metropolitan New York firefighters, almost all of whom had worked as firefighters for at least twenty years, forty-nine percent had abnormalities on chest x-ray that are characteristically caused by prior exposure to asbestos. It was concluded that firefighters are at risk for scarring of the lungs and pleura due to occupational asbestos exposure. It was thought that much of that exposure occurred during the overhaul phase of firefighting [107].

It is clearly established that asbestos causes mesothelioma and lung cancer in humans [160, 76]. IARC has classified asbestos as a Group 1 carcinogen for which the evidence is sufficient [79]. The Panel has previously reported that it can also cause laryngeal and gastrointestinal cancers [77, 160]. It is also carcinogenic to animals [79].

Asbestos is a designated substance in Ontario. Regulations limit most workers' exposure to asbestos in workplaces, construction projects and in buildings and repair operations [142, 143].

Benzene

Benzene may be present at fire sites where it is being used as an ingredient for the manufacture of various products (e.g. medicinal chemicals, dyes, artificial leather, linoleum, oil cloth, varnishes and lacquers) and as a solvent for waxes, resins and oils [20]. It is also a common decomposition product of many organic materials.

After carbon monoxide, benzene is generally the second most commonly found organic constituent of fire smoke, typically present in high concentrations in the fire environment [19]. Mean airborne concentrations ranging from 28-63 ppm of benzene were measured from grab samples collected at various content/building or car fires. These concentrations were two to four times Ontario's current maximum allowable concentration of 15 ppm at any time [144]. The measurements from individual air samples were

as high as 16 times this maximum allowable concentration (ranging from not detectable to 250 ppm). These findings are similar to previous reports of benzene measurements at fire sites which reached levels greater than 150 ppm [198].

A more recent study showed that firefighters could be highly exposed to benzene even with the use of self-contained breathing apparatus. Levels as high as 21 ppm were measured from air samples collected from inside SCBA masks worn at fire sites [82].

In addition to its irritative and narcotic effects, benzene damages the bone marrow resulting in reduced red and white blood cells and platelets. This can lead to anemia, susceptibility to infection and clotting disorders [20]. It is also known to produce both non-malignant tumours and leukaemia in rats and cause mutation and DNA damage in rodent cell cultures [79].

IARC has classified benzene as Group 1, for which there is sufficient evidence of carcinogenicity to humans (causing several types of leukaemia) and to animals (causing cancer of multiple sites). It has also produced both non-malignant tumours and leukaemia in rats and caused mutation and DNA damage in rodent cell cultures. Lymphoid cancer has been induced in mice by inhalation of benzene [79].

Benzene is a designated substance in Ontario [144].

Carbon monoxide/dioxide

Carbon monoxide and carbon dioxide are common occupational exposures of firefighters because they are natural products of combustion and are necessarily present at every fire. There are very different health effects associated with exposure to these chemicals either separately or combined..

Mean carbon monoxide concentrations measured from grab samples ranged from 22.7 ppm (auto fire), 235 ppm (content fires) to 272 ppm (building fires) [19], all of which were well below the current Ontario STEV of 400 ppm; however, a much higher mean of 500 ppm was reported in another study [5]. The difference may be due to the nature and intensity of the fires studied, as well as the sampling duration and location selected.

Studies using rats exposed to varying concentration ratios of carbon monoxide and carbon dioxide showed that carbon monoxide in the presence of 5% carbon dioxide is twice as toxic as carbon monoxide alone. Apparently, carbon dioxide increases the absorption of carbon monoxide and prolongs its effects [30].

Unlike carbon monoxide, carbon dioxide is a simple asphyxiant. It is also considered a potent stimulus to respiration, as well as being both a depressant and an excitant of the central nervous system [154].

No mean concentration was reported for carbon dioxide in any of the available studies. Grab sample measurements reported in several studies ranged from below 1,000 ppm to 60,000 ppm (a difference of at least 60 times), the latter being twice the STEV of 30,000 ppm for carbon dioxide. However, such levels may be uncommonly high for carbon dioxide at most fires. A recent study of various fires reported carbon dioxide levels to be 300-5410 ppm during the knockdown phase and 130-1420 ppm in the overhaul phase [82].

When inhaled, carbon monoxide is quickly absorbed into the blood stream and binds with red blood cells to form carboxyhemoglobin. This complex displaces oxygen on red blood cells by a factor of 200 times and interferes with the transfer of essential oxygen to body tissues [60, 205, 222]. Carbon monoxide is directly toxic to the heart [20] and may be involved in the development of atherosclerosis [222]. Atherosclerosis may predispose an individual to aortic aneurysm [158].

Mean carboxyhemoglobin levels of about 4% were measured in non-smoking firefighters exposed to carbon monoxide concentrations as low as 200-1000 ppm [55]. These levels approach those in smokers (5-7%) who consume an average of 1.5 packs of cigarettes per day [180].

The maximum concentration of carbon monoxide in samples measured typically exceeded 1,000 ppm [19, 53, 82] and sometimes reached potentially lethal levels of 3,000 ppm [5] to 5,000 ppm [198]. Levels measured during the knockdown phase (up to 1900 ppm) were substantially higher than during the overhaul phase (up to 82 ppm) [82].

Carbon monoxide may also worsen the damage to hearing caused by noise [46, 223]. Occupational hearing loss among firefighters will be discussed in detail in a subsequent Panel Report.

Chloroform (trichloromethane)

Chloroform may be found as a constituent of solvents and as a decomposition product of organic materials in fires.

Chloroform has been quantified in the fire environment, but at relatively low concentrations [112].

It is known as a skin and eye irritant [20], a central nervous system depressant and a cause of liver and kidney damage [149]. Cardiovascular and carcinogenic effects in humans are generally not known. However, IARC did report sufficient evidence that chloroform is carcinogenic to animals (Group

2B), causing cancers of the liver and kidney [79]. This substance can also cause genetic damage and is a reproductive toxin in rodents [20].

Diesel exhaust

Diesel exhaust is a complex mixture which includes polycyclic aromatic hydrocarbons (PAHs), benzene, formaldehyde, etc. There is to date no single exposure standard or guideline available for diesel exhaust exposure. The combined particulate fraction of diesel exhaust is usually determined as total particulates and as methylene chloride extract of the total particulate fraction. The latter is used as an indicator of the content of PAHs.

A survey of 23 Ontario fire stations concluded that firefighters may be significantly exposed intermittently to diesel exhaust in the fire station from the operation of diesel-powered vehicles. Carbon monoxide levels were used as a marker for diesel exhaust concentration. For purposes of comparison, carbon monoxide concentration was measured directly from the vehicle exhaust and averaged about 200 ppm (ranging from 60 to 750 ppm). Carbon monoxide levels were found to be less than 5 ppm (background level) in the fire stations but were higher than 50 ppm (up to 120 ppm) on the apparatus floor when vehicles were started or returned to the station. Levels as high as 33 ppm were measured in the living quarters of the fire station during these two periods [97].

In most cases, carbon monoxide measured on the apparatus floor decreased to levels below 10 ppm within ten minutes after reaching peak concentration. This was largely attributed to the different types of control measures used in fire stations, such as mechanical tailpipe exhaust, structural barriers, natural and mechanical ventilation, and pole hole location [97].

A union representative of firefighters advised the Panel that in some fire stations, it is common practice to regularly start the vehicle engines while they are inside the station to ensure that they are in working order. Diesel exhaust exposure could also be significant when, for example, only one vehicle in a multi-vehicle station responds to a call. In such a circumstance, the vehicle is started indoors and those firefighters who remain in the station are exposed. The Panel was advised that the walls and floors in most fire stations are washed every year, but in some cases the walls become "black" with diesel exhaust particulate within about six months. Most but not all urban stations have adequate separation between living quarters and the apparatus floor [28].

IARC has classified diesel engine exhaust as probably carcinogenic to humans, particularly in the cases of lung and bladder cancers (Group 2A). It reports sufficient evidence that diesel exhaust causes lung cancer in animals [80].

Formaldehyde

Formaldehyde is used in the manufacture of resins, textiles, embalming fluids, fungicides, air fresheners [20], plastics, adhesives, wood products, insulation, paints, leather and rubber [164]. It may therefore be present as a decomposition product in fires involving such materials.

Brandt-Rauf (1988) studied different types of fires and reported mean (grab sample) formaldehyde concentrations of 0.8 ppm and 0.5 ppm in the air at content fires and building fires, respectively. Airborne formaldehyde was not detected in the automobile fires observed. These means are two to four times below Ontario's STEV of 2 ppm for formaldehyde in workplace air, although levels as high as 3.3 ppm and 8.3 ppm were measured in content fires and building fires, respectively [19]. Thus, formaldehyde levels which could cause acute health effects in humans may be present in certain areas or during certain phases of a fire.

In another study, formaldehyde levels as high as 8 ppm were measured during the knockdown phase of the fires, as well as a maximum level during the overhaul phase of 0.4 ppm [82].

Formaldehyde is a primary irritant to the mucous membranes of the eyes, nose and respiratory system and can cause headaches, cough, difficulty sleeping, diarrhoea, nausea, phlegm, weakness, vomiting, dizziness, wheezing, chest pain and tightness, breathlessness, rash, bronchitis and pneumonia [196]. Acute respiratory tract irritation can lead to pulmonary edema and pneumonitis [131].

According to IARC, formaldehyde is probably carcinogenic (Group 2A) [79]. Excess cancer rates occurred in more than one study for the following cases: Hodgkin's disease, leukaemia, and cancers of the buccal cavity and pharynx (particularly nasopharynx), lung, nose, prostate, bladder, brain, colon, skin and kidney [79].

An excess of deaths from lymphopoietic cancer was found among formaldehyde-exposed workers by Levine et al. (1984).

Thomas and Waxweiler report an association between occupational exposure to formaldehyde and brain cancer [191]. The incidence of brain cancer was consistently elevated among professional groups (embalmers, pathologist, anatomists) but not among industrial workers exposed to formaldehyde. There was correlation between these professionals' years of exposure and brain cancer [14]. For the pathologists studied, chronic exposure to other substances such as organic solvents, tuberculosis infection and drugs have also been implicated [64].

Halons

Halons are fire extinguishing agents. Halon fire extinguishers are present in offices, factories, public buildings, etc. [20].

As a rule, Halon gases are not lung irritants except at high concentrations. The most important toxicological effects of Halons are on the central nervous system (CNS) and on the cardiovascular (CV) system. Clinically important CNS effects almost always appear at lower levels of exposure than do CV effects. CNS effects resulting from Halon overexposure are: alterations in perception, increase in reaction time, and reduced ability to concentrate on complex intellectual tasks [197].

The cardiovascular effects of Halons are the most significant hazard of their use. They can cause decreased blood pressure and cardiac arrhythmias. Halons appear to interact with endogenous catecholamines (chemical messengers in the body) such as adrenalin. It is thought that Halons sensitize the heart to the arrhythmogenic action of adrenalin. This assumption is based on the supposed release of adrenalin from the adrenal medulla during excitement, fear or other stressful stimuli such as that experienced by firefighters. Exposure to other toxicants such as chloroform in fires which can also cause similar effects would potentiate the cardiac effects of Halons [20].

Hydrogen chloride

Hydrogen chloride is used in the manufacture of pharmaceuticals, chlorine, vinyl chloride and alkyl chlorides, and in the chlorination of rubber [20]. Hydrogen chloride gas is one of at least 75 identifiable potentially toxic compounds produced by the combustion of polyvinyl chloride [40]. Since polyvinyl chloride is widely used in home construction, furnishings, electric wire, telephone cables, office equipment [40] and wallcoverings [30], hydrogen chloride is likely to be present in most fires [2, 112].

In a study of different fires, mean hydrogen chloride concentrations of 0.1 ppm and 3.3 ppm (grab samples) were measured in the air of building and content fires, respectively [19]. These concentrations are below Ontario's CEV of 5 ppm [143]. However, levels that exceeded twice that CEV (ranging from not detectable to 13.3 ppm), were measured in some samples collected from content fires. Other studies reported similar and even higher findings, with levels as high as 40 ppm [103], 150 ppm [43] and 200 ppm [198] of hydrogen chloride at sites of "non-specified mixed" fires.

Acute effects of hydrogen chloride exposure include eye, skin and throat irritation [106], as well as impairment to respiratory functions [30]. It has also been found to have cardiotoxic effects in rats [138].

Hydrogen cyanide

Hydrogen cyanide is commonly used in the production of chemicals such as resin monomers, cyanide salts, nitriles (e.g. acrylonitrile) [22] as well as rodenticides and insecticides [20]. It is produced by the incomplete combustion of both natural fibres (such as wool and silk) and synthetic polymers (such as polyurethane, polyacrylonitrile, nylon and melamine) widely used in building materials and furnishings [175].

While one study reported hydrogen cyanide in only 10-15% of the fires surveyed [103], others have detected it in as many as 47% of the fires studied [112]. Since polyvinyl chloride is a pyrolysis product of polyvinyl chloride, which is a very widely used polymer, hydrogen cyanide is likely to be involved in most fires [2].

Mean or average concentrations of hydrogen cyanide reported in different studies ranged from 0.04 [53] to 3.7 ppm [103] and from 2.9 to 15 ppm [19]. The highest level was reported for content fires and exceeded the CEV of 10 ppm [143]. The measurements from such fires ranged from below detection to 75 ppm [19], a level five times the CEV.

Hydrogen cyanide is much more potent and faster acting than carbon monoxide [2] and can be rapidly fatal [30]. Both hydrogen cyanide and carbon monoxide are chemical asphyxiants that render the body incapable of using an adequate supply of oxygen. While carbon monoxide interferes with the transport of oxygen to the tissues by its affinity to haemoglobin, cyanide alters the cellular use of oxygen in energy production [30]. Carbon monoxide and cyanide are additive in producing changes in blood flow within the brain but may act synergistically on cerebral metabolism, so that their combined effects are greater than that expected by either substance alone [153].

Nitrogen dioxide

Nitrogen dioxide is a common decomposition product of fires. It may also be present in fires which occur where it is being used as an ingredient in the manufacture of chemicals [20], nitric and sulphuric acids, and explosives.

A mean concentration of 0.47 ppm was reported for nitrogen dioxide in 8-minute air samples collected at non-specified mixed fires [53]. This concentration is well below the STEV of 5 ppm set for nitrogen dioxide in Ontario workplaces [143]. In another study, nitrogen dioxide levels as high as 10 ppm [198] were observed in some of the samples, although no mean values were reported.

Nitrogen dioxide is a strong lung irritant and can cause pulmonary edema [20]. NIOSH considers it a suspected carcinogen based upon animal studies and limited epidemiologic evidence [149].

Organic solvents

Organic solvents are widely used in various workplaces to dissolve other organic materials and are classified into several broad categories[5].

Firefighters may be exposed to a complex mixture of organic solvents present in fire stations, at fire sites, and from decomposition products of materials involved in the fires.

After carbon monoxide, benzene is generally the second most commonly found organic constituent of fire smoke [19]. In a review of various studies, grab sampling measurements of other solvents such as chloroform, dichlorofluoromethane, methylene chloride, perchloroethylene, toluene and trichloroethylene were well below the respective exposure limits used for comparison by the authors. These limits are comparable to the STEVs or TWAEVs prescribed for the individual chemical agents mentioned.

All organic solvents affect the central nervous system to some extent, acting as depressants and anaesthetics. They also cause dermatitis and other health effects depending on the solvent and the route and extent of exposure. These effects range from narcosis to death from respiratory arrest. While some (e.g. n-hexane and methyl n-butyl ketone) cause peripheral neuropathy when chronically exposed, others (e.g. carbon tetrachloride) are recognized more for their acute injuries to the liver, kidneys and gastrointestinal tract [81]. Exposure to trichloroethylene, a commonly used solvent, has been found to cause liver cancer in mice [154]. Benzene causes considerable adverse effects on the blood-forming tissues and bone marrow (see "Benzene", above). Carbon disulphide has been linked to cardiovascular disease [84]. There is evidence to suggest that solvent exposure can also exacerbate the damage to hearing caused by excessive noise [168].

Polycyclic aromatic hydrocarbons (PAHs)

PAHs are multi-ring aromatic compounds found widely dispersed in nature. They are formed during the combustion of many organic materials (for example, diesel fuel) and high-temperature processing of crude oil, coal and coke. They also occur in tobacco smoke and grilled, smoked and fried foods [81].

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5 They are: **aliphatic hydrocarbons** (hexane, benzine, mineral spirits); **cyclic hydrocarbons** (cyclohexane, turpentine and kerosene); **nitro-hydrocarbons** (nitroethane); aromatic hydrocarbons (benzene, toluene and xylene); **alcohols** (methanol and ethanol); **glycols** (ethylene glycol); **esters** (ethyl acetate); **ketones** (acetone); **aldehydes** (acetaldehyde); **ethers** (ethyl ether) and **halogenated hydrocarbons** (trichloroethylene and carbon tetrachloride).

Only one study reported concentration of airborne PAHs in fire smoke. The measurements ranged from below detection to 0.5 mg/m^3 during knockdown and below detection to 0.02 mg/m^3 during overhaul. Personal samples from firefighters showed no measurable exposure to PAHs when the SCBA was worn [82].

No short-term exposure limit has been prescribed in Ontario for PAHs *per se*, although a TWAEV of 0.2 mg/m^3 has been set for coal tar pitch volatiles (total benzene-solubles). As already mentioned, a comparison of the aforementioned measurements with a TWAEV may not be meaningful because of the short-term or intermittent exposure profile of firefighters to contaminants in fire smoke.

In the human body, PAHs are metabolized to more water soluble compounds which are excreted through urine or bile. While many PAHs, such as naphthacene and anthracene, are not known to be carcinogenic, others (eg. benzo(a)pyrene, benzo(a)anthracene and pyrene) and their metabolites have shown to be slight to potent carcinogens. These PAHs, particularly benzo(a)pyrene, may be linked to increased risk of cancer of the lungs, colon, pancreas, stomach, pharynx and bladder reported in petroleum refinery workers and in workers exposed to coke, coal tar pitch and asphalt. Tar and pitch exposure is associated with benign and malignant skin tumours [81, 79].

A detailed review of the epidemiological literature by Thomas and Waxweiler identified an association between PAHs and brain cancer (1986). Other authors have suggested a link between exposure to PAHs and leukaemia [62] and cancers of the bladder, kidney and ureter [59].

Soots

Since all fires create soots, it is likely that firefighters are also significantly exposed during firefighting. Soots contain polycyclic aromatic hydrocarbons (PAHs), many of which are known carcinogens in humans. These particulates have been measured in detectable amounts in the smoke of building fires [82].

IARC reports that there is sufficient evidence to establish soots as carcinogenic to humans (Group 1) in the cases of skin, scrotal and lung cancer. Statistically significant excesses in mortality from esophageal and liver cancer and leukaemia were also found among chimney-sweeps exposed to soots [79].

Vinyl chloride

Vinyl chloride is used primarily to manufacture plastic articles (from polyvinyl chloride) such as building and construction materials (pipes, ducts,

floor tiles, electrical wire and cable), packaging (films, sheets, bags and bottles), clothing, insulation, automobile upholstery and mats, records, toys and a variety of consumer goods [21]. When sufficiently heated, these articles can release vinyl chloride and other products of decomposition which are health hazards. In all, at least 75 potentially toxic products have been identified in the thermal degradation of polyvinyl chloride [40].

The Panel is aware of only one study by Markowitz et al (1989), which reported measurements of vinyl chloride in fire smoke. Only a small amount of vinyl chloride was detected in the smoke from the decomposition of plastics. Measurements from a fire involving polyvinyl chloride showed that the products of pyrolysis consisted mostly of nitrogen, hydrogen, carbon monoxide and hydrogen chloride [106]. Also identified in lesser amounts were carbon dioxide, hydrogen cyanide, benzene and methane. The concentration of vinyl chloride itself, however, was below the limit of detection (less than 0.2 ppm) in that study.

Short-term exposures to high concentrations of vinyl chloride have resulted in euphoria, dizziness, respiratory irritation, headache, nausea, irritability, poor memory and tingling sensations [20]. A high incidence of Raynaud's syndrome, hypertension and coronary deficiencies had also been reported in workers exposed for at least 5 years [90].

According to IARC, there is sufficient evidence (Group 1) that vinyl chloride is carcinogenic, causing cancers of the liver, brain, lung and haematolymphopoietic system in humans. IARC also noted findings of excessive melanoma, gastric and gastrointestinal cancers in exposed individuals [79]. Several authors also reported an association between vinyl chloride and brain tumours, particularly gliomas [191, 93]. It has also shown to be carcinogenic, mutagenic and genotoxic in studies on rodents and cell cultures [79].

Vinyl chloride is a designated substance in Ontario [141].

Other

Methylene chloride and sulphur dioxide have also been identified at fire sites. They cause eye and skin irritation but are not known to lead to the major health outcomes upon which the Panel has decided to focus in this Report.

SUMMARY

There is evidence that many of the chemical substances to which firefighters may be exposed are carcinogenic to humans and animals. Some of these substances cause cardiovascular, respiratory and central nervous system effects. In addition, carbon monoxide and solvent exposure may worsen the damage to hearing caused by excessive noise exposure.

- **Other hazards of firefighting**

The physical activities demanded by their work may also affect firefighters' cardiovascular health.

In 1992, a York University research team published a detailed study of the activities involved in firefighting [51]. Based upon that information, they developed a fitness screening protocol for firefighter applicants [52] which has recently been adopted by several fire departments.

This research identified the following physical tasks which are commonly required during active firefighting:

- carrying equipment up stairs in high-rise buildings while wearing breathing apparatus and turnout gear, which together weigh 48.4 pounds dry and are heavier when wet;
- advancing charged hoses, sometimes from a distant hydrant, outdoors around obstacles or in icy conditions and indoors through hallways or stairs;
- carrying heavy equipment long distances from the truck to a fire site, particularly when garbage and furniture interfere with access to the fire site;
- breaking down doors, walls, ceilings and roofs; forcible entry through walls or steel security doors using hand tools;
- raising ladders, sometimes with an insufficient number of firefighters; using an axe while on a ladder;
- working overhead with a pike pole or other equipment; for example, breaking through a roof while on a ladder;
- rescuing victims from a roof or window using a ladder, or from confined areas using hand and power tools; moving victims from damaged cars or collapsed buildings;
- raising and lowering equipment or victims from high-rise windows using ropes;
- fighting fires for extended periods of time; conducting lengthy extrication and rescue operations, for example, in multi-vehicle accidents, industrial fires, train derailments, etc.; and working overhead or in awkward positions for extended periods of time [51].

A firefighter's endurance is reduced by self-contained breathing apparatus and other protective clothing which is hot, cumbersome and heavy [179]. The extra weight and effort causes more rapid breathing which is made even more difficult by using the respirator [51]. As mentioned above, the heavy exertion demanded by fighting a fire causes more rapid and deeper breathing which increases delivery of toxins to deep within the lungs [60].

In older buildings composed of heavier construction materials, fires reach higher temperatures and spread more rapidly. Firefighters must work more quickly, encumbered by these physical constraints and in even higher heat [51].

Fighting high-rise building fires requires climbing several flights of stairs carrying the weight of protective gear, tools and hoses, and removing heavy windows or opening concrete walls. Newer buildings often involve concrete construction and toxic materials [51]. Concrete absorbs heat and toxins, then releases them gradually as it cools [51, 60].

Obviously, firefighters are exposed to very high temperatures. Heat stress is compounded by the insulating properties of the protective clothing and by physical exertion, which results in endogenous heat production [60]. Research indicates that heat stress contributes to ischemia [6] and to the risk of a myocardial infarction in a predisposed individual [126].

In addition to burns, radiant heat may cause skin injuries such as erythema and telangiectasia [60].

Firefighting in winter conditions can pose special problems as water freezes and firefighters undergo large fluctuations in body temperature when they repeatedly enter and exit the fire site [51].

Darkness and smoke decrease visibility, impede search and firefighting procedures and may lead to accidents and traumatic injuries [51].

High noise levels make firefighting more difficult and dangerous. Noise interferes with speech and hazard communications and drowns out warning signals, which can eliminate firefighters' ability to prevent traumatic injuries. Loud noise can also cause stress reactions which are measurable in increased heart rate and elevated blood pressure [110] and some studies have documented noise-induced hearing loss among firefighters [159].

Firefighters routinely face unknown personal danger and, unlike most other workers in Ontario, they have no legal right to refuse work which is unsafe[6]. They are often responsible for the safety of other people. These

[6] Section 23(2), *Occupational Health and Safety Act*, R.S.O. 1980, chapter 321 as amended.

responsibilities, quite understandably, cause high levels of psychological stress [60, 16].

Finally, there is some evidence that shiftwork has a negative effect on subjective, and to a lesser extent, objective measures of health. Normal 24-hour (circadian) rhythms are disturbed by shiftwork, which can interfere with sleep patterns. Family and social activities may also suffer [50].

b) Considerations for interpreting epidemiological evidence

(i) Selected terms[7]

Dose-response:

A dose-response trend is shown when an increase in the "dose" (exposure level, intensity or duration) corresponds to an increase in the "response" (death or disease). In the studies of firefighters, dose is usually measured by duration of employment.

Latency period:

The period of time between first exposure to a substance(s) and the appearance of the disease which it has caused. In the studies discussed below, latency is usually measured by time since first employment.

Standardized Mortality Ratio (SMR):

An SMR is computed by comparing the number of deaths **observed** (i.e. that *occurred*) among firefighters with the number of deaths which are **expected** based upon a comparison group of the same age and sex, during the same time period:

$$\text{SMR} = \frac{\text{"observed" deaths among firefighters}}{\text{"expected" deaths}} \times 100$$

Most authors multiply the ratio by 100, but some do not. An SMR greater than 100 (or 1) suggests an excess risk of death. Epidemiologists evaluate the ***statistical significance*** of an elevated SMR by using the *95% confidence interval*, which is the range in which the true SMR would fall 95% of the time.

If the lower end of the 95% confidence interval is **above** 100 (or 1), the likelihood that the excess mortality is due to chance is less than 5% (or 1 out of 20). If the SMR is **under** 100 (or 1) and the upper 95% end of the confidence interval is under 100 (or 1), a statistically significant **decrease** in deaths is indicated, compared to the number of deaths that would normally be expected.

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[7] Readers may also wish to review "Appendix B" which includes an explanation of additional terms and abbreviations used in this Report.

(ii) Potential sources of bias

When evaluating epidemiological evidence, it is important to consider the following factors.

Ascertainment bias

The results of epidemiological studies can sometimes be skewed by an *ascertainment bias.* This may occur when the total cohort is not (or usually, cannot be) traced in order to identify illness or death; in other words, they are "lost to follow-up". In order to err on the side of caution, epidemiological studies assume that subjects lost to follow-up are alive. This means that they contribute "person-years" throughout the study period even if they have died. This has the effect of overestimating the time subjects worked and remained healthy as well as underestimating the "observed" number of deaths. If the outcomes for those lost to follow-up *were* known, their deaths would raise the mortality rates in one or more categories of disease, because death is a common reason for the inability to trace members of a cohort.

For example, in a study by Kusiak et al. (1993) of lung cancer among uranium miners, Social Insurance Numbers (SINs) were available for only 63% of the men in uranium mines (largely because SINs were not issued until 1965). The SMR for those with SINs was 225, whereas for those without SINs, it was only 135. The authors indicated that the "additional identifying information obtained from the SIN Registry permitted the identification of a much higher proportion of the deaths of Ontario miners." According to the calculations of the IDSP, improved ascertainment contributed significantly to a 67% increase in SMRs when the vital status of miners with SINs was compared to the vital status of miners without SINs. (This may also be partly due to differences in exposure and follow-up between the two groups.)

Whenever less than 100% of the cohort is traced, an ascertainment bias will cause an underestimation of risk, unless the author specifically adjusts for this bias. The larger the number of subjects who are not traced, the larger the underestimation of risk.

The Panel reviewed the percentage of subjects whose health outcomes were traced in each firefighter cohort study discussed in this Report and found that all but one study traced at least 90% of the members of each cohort.

Proportion of subjects traced in firefighter mortality studies:

Beaumont et al., 1991	97%
Demers et al., 1992	98%
Eliopulos et al., 1984	97.3%
Guidotti, 1993	96%
Hansen, 1990	over 99%
Heyer et al., 1990	95.4%
L'Abbé & Tomlinson, 1992 (IDSP)	97.8%
Lewis et al., 1982	95%
Mastromatteo, 1959	78.4%
Musk et al., 1978	92.1%
Tornling et al., 1994	100%
Vena & Fiedler, 1987	97.5%

As shown above, in one very early study [109] 21.6% of firefighters were lost to follow-up which could introduce ascertainment bias. Nevertheless, that study identified statistically significant increases in cardiovascular-renal deaths among Toronto firefighters. Its findings were probably one of the reasons for the subsequent emphasis and concern about the risk of cardiovascular disease among firefighters.

The healthy worker effect

Many epidemiological studies compare workers to the general population. Since the general population includes people who do not or cannot work due to illness or disability, *including work-related disability*, a working population is usually healthier overall and usually has a lower rate of mortality for most causes of death. The influence of these factors on the results of epidemiological studies is known as the *healthy worker effect*. This effect results in lower SMRs than would occur if comparisons had been made with a working population.

Almost all authors of firefighter studies commented that the healthy worker effect had influenced the cardiovascular findings. The influence of the healthy worker effect on findings for cancer, however, is somewhat controversial.

There is no screening test for susceptibility to cancer, but some experts believe that workers who are selected for being at low risk for cardiovascular and respiratory disease are healthier overall and are therefore less likely to develop cancer [111, 184]. Others think that if those who are susceptible to heart and lung disease are excluded from the fire service, the ones who are admitted will have a statistically higher chance of developing cancer [155]. Monson's review of studies of ten groups of workers concluded that the healthy worker effect had little or no effect on cancer mortality [125]. Alternatively, Sterling and Weinkam concluded that a healthy worker effect

amounting to 20% to 40% reduced mortality was shown to persist over the entire age range for various causes including cancers [184].

The IDSP, in its Report Number 3 (July, 1988), concluded that the healthy worker effect must be taken into account when interpreting epidemiological studies of mortality or morbidity from any cause, including cancer. No uniform correction factor should be used because each study requires individual interpretation.

Comparing working populations to the general population

When an SMR is computed, the general population, on which the expected number of deaths is based, includes the observed number of deaths from the study population. This is because these deaths contribute to both populations, that is, the deaths are counted for both the observed and the expected. For example, deaths due to nasal cancer are almost always caused by occupational factors. An SMR for nasal cancer will thus be underestimated.

Multiple tests of significance

This refers to the possibility that when numerous disease outcomes are examined, statistically significant associations will be found for some of them by chance alone.

The survivor effect

Because adequate health is required for continuing employment, workers with cardiovascular disease may leave firefighting service, or transfer to positions where they do not encounter these exposures. It is likely that only the healthiest workers continue to be employed for a long term. The influence of this phenomenon is known as *the survivor effect*.

Since duration of employment was the only way that exposure could be estimated for most firefighter studies, a survivor effect would obscure a dose-response relationship. That is, a correlation between the length of exposure and illness would be reduced by the fact that those with the longest exposure would mainly be the healthiest workers [86].

c) The IDSP Mortality Study of Firefighters in Metropolitan Toronto

The purpose of the IDSP study was to examine mortality by cause of death for firefighters in Metropolitan Toronto, with particular emphasis on cardiovascular diseases.

This original research was undertaken because a literature review conducted for the Panel found too few studies to provide answers about the chronic health effects of firefighting. There was, however, some evidence to "suggest that it is biologically plausible that firefighting could lead to the development of respiratory, cardiovascular and neoplastic disease" [222]. While the Panel has considered all the available evidence, it has chosen to highlight this study because its subjects are Ontario workers, the jurisdiction for which the IDSP is responsible for making policy recommendations. The IDSP study is one of the largest studies of mortality among firefighters ever conducted.

In this study, a Standardized Mortality Ratio (SMR) of more than 100, with the lower end of the 95% Confidence Interval exceeding 100, was interpreted by authors Kristan A. L'Abbé and George A. Tomlinson as a *statistically significant* increase in firefighter deaths. Ontario males in the same age groups, and whose deaths occurred during the same time periods, were used to make these comparisons.

Name, sex, date of birth, date of hire and termination, reason for termination (where available), job titles and assignments, social insurance and employee numbers were obtained from employment records kept by all six Metropolitan Toronto fire departments (the Cities of Toronto, North York, Scarborough, Etobicoke and York, and the Borough of East York).

The size of the cohort was 5995 before exclusions. Since there were only 146 females, they were excluded. Also excluded were those who were aged 85 or more or who had died before the study period began on January 1, 1950 and those for whom vital information was unavailable. There were 5414 males with six months or more employment as firefighters in the cohort for the principal analysis, which included 777 deaths. The analyses which used duration of employment included fewer, 5373 firefighters including 753 deaths, because some who were not currently employed had no known termination date [86].

Identifying information was linked by Statistics Canada with records from the Canadian Mortality Data Base to identify each cause of death. Any information which might identify individual firefighters was removed from the data returned to the IDSP by Statistics Canada.

The principal tables from the Scientific Report are reproduced below. Table 2 shows the overall SMRs, Table 3 shows SMR by duration of employment, Table 4 indicates SMRs by years since first employment as a firefighter and Table 5 lists SMRs by age.

As shown, the study identified statistically significant increases in mortality rates for brain and nervous system cancers and for other malignant neoplasms (including largely secondary or unspecified types) among Toronto firefighters. Aortic aneurysm mortality was also increased to a statistically significant degree, but other cardiovascular causes of death were not comparably elevated. The rate of death from gallbladder diseases was elevated but did not reach statistical significance overall.

The Panel cautions against complete reliance upon these findings because the statistically significant increase in "other malignant neoplasms" probably reflects increases in cancers whose primary type was not identified. This would tend to underestimate the reported rate of cancer in some of the other categories.

In their original work, L'Abbé and Tomlinson included deaths which occurred during the first five years of exposure to firefighting. Since it is generally recognized that it takes a minimum of five years after first exposure to a carcinogen for cancer to develop [111], the Panel was concerned that including deaths which occurred during the five years after first exposure could be misleading [75]. To examine this possibility, the Panel asked Mr. Tomlinson to re-analyze the cancer data after eliminating deaths and person-years during the five years since first employment. That analysis showed that, since there were so few cancer deaths within five years of first employment, the SMRs for these analyses are very similar to those for the complete analysis [193].

It might have been informative to analyze causes of death by municipality to see whether any trend in disease patterns exists; however, that was not possible for several reasons. First, data about the number of fires in each municipality, as distinguished from the number of other calls, has been compiled by Ontario's Office of the Fire Marshall only since 1983 [135]. Firefighters routinely transfer within different platoons and fire halls. The number of fires attended by individual firefighters during the period covered by the study (1950-1989) was not available from any of the Fire Departments studied [113, 123, 148, 156, 182]. For all these reasons, it was impossible to conduct more detailed analyses and, in any event, the breakdowns required to do so would involve numbers too small to provide reliable data. More general information from published literature about other jurisdictions is, therefore, the best exposure evidence currently available to the Panel.

Most of the other studies of firefighters' mortality have had insufficient exposure information to conduct such an analysis. Some researchers have

attempted to design other ways to approximate firefighters' exposure to fire and chemical hazards [59, 8, 129, 195]. Since there is usually insufficient information about job assignments and activity levels, most studies have had to use duration of employment as a crude measure of exposure [86].

TABLE 2
Mortality by Detailed Cause for Total Cohort, IDSP Mortality Study of Fire Fighters in Metropolitan Toronto (L'Abbé and Tomlinson, 1992)

Cause of death (ICD 9)	OBS.	EXP.	SMR	95% C.I.
ALL CAUSES (000-999)	777	823.50	94	(88-101)
INFECTIVE, PARASITIC DISEASES (1-39)	1	7.21	14	(0- 77)
ALL MALIGNANT NEOPLASMS (140-208)	199	190.39	105	(90-120)
Pharynx (146-149)	4	2.87	139	(38-357)
Oesophagus (150)	2	5.04	40	(5-143)
Stomach (151)	7	13.79	51	(20-105)
Colon (153)	11	18.25	60	(30-108)
Rectum and rectosigmoid junction (154)	13	7.60	171	(91-293)
Liver and bile ducts (155)	2	2.37	84	(10-305)
Pancreas (157)	14	9.98	140	(77-235)
Larynx (161)	1	2.70	37	(1-206)
Trachea, bronchus & lung (162)	54	57.08	95	(71-123)
Malignant melanoma (172)	2	2.76	72	(9-262)
Prostate (185)	16	12.09	132	(76-215)
Testis (186)	3	1.22	246	(51-719)
Bladder (188)	7	5.49	128	(51-263)
Kidney and ureter (189)	2	4.63	43	(5-156)
Brain and other nervous system (191-192)	14	7.00	200	(109-336)
Lymphatic & haematopoietic tissue (200-208)	18	18.39	98	(58-155)
Lymphosarcoma (200)	3	1.48	203	(42-592)
Hodgkin's disease (201)	1	2.19	46	(1-254)
Multiple myeloma (203)	1	2.59	39	(1-215)
Lymphatic leukaemia (204)	4	2.11	190	(52-485)
Myeloid leukaemia (205)	4	3.35	119	(33-306)
Other	5	6.67	75	(24-175)
Other malignant (195-199)	20	7.08	282	(172-436)
ENDOCRINE, NUTRITIONAL DISEASES (240-279)	5	14.61	34	(11- 80)
CIRCULATORY SYSTEM (390-459)	384	387.79	99	(89-109)
Ischemic heart disease (410-414,429.2)	289	278.38	104	(92-117)
Acute myocardial infarction (410)	205	191.65	107	(93-122)
Cerebrovascular disease (430-438)	41	54.02	76	(55-103)
Arteriosclerosis (440-448)	24	16.98	141	(91-210)
Aortic aneurysm (441)	19	8.40	226	(136-353)
Diseases of veins, etc. (415, 451-459)	8	4.78	167	(72-330)
RESPIRATORY SYSTEM (460-519)	45	46.12	98	(71-131)
Chronic bronchitis, emphysema, asthma (490-493, 496)	28	21.47	130	(87-189)
DIGESTIVE SYSTEM (520-579)	45	36.85	122	(89-163)
Gallbladder diseases	5	1.69	296	(96-690)
GENITOURINARY SYSTEM (580-629)	11	10.41	106	(53-189)
SYMPTOMS/ILL-DEFINED (780-799)	1	6.06	17	(0- 92)
EXTERNAL CAUSES (E800-999)	70	100.72	69	(54- 88)
Motor vehicle traffic accidents (810-825)	22	31.10	71	(44-107)
Accidental falls (880-888)	9	7.82	115	(53-219)
Accidents caused by fire & flames (890-899)	4	3.23	124	(34-317)
Suicide (950-959)	18	26.68	67	(40-107)
OTHER KNOWN CAUSES	16	22.81	70	

(shading included for reading ease only)

TABLE 3: Standardized Mortality Ratios by Duration of Employment for Selected Causes, IDSP Mortality Study of Fire Fighters in Metropolitan Toronto (L'Abbé and Tomlinson, 1992)

	Duration of Employment								
	< 15 Years			15-29 Years			≥ 30 Years		
Cause	Obs.	SMR	95% CI	Obs.	SMR	95% CI	Obs.	SMR	95% CI
All Causes	130	88	74-104	240	97	85-110	383	91	82 101
All Cancers	36	137	96-190	53	87	65-114	100	100	81-122
-pharynx	1	227	6-1265	0	0	*******	3	236	49-699
-rectum	0	0	*******	5	220	71-513	8	180	78-355
-pancreas	2	172	21-621	3	92	19-269	9	165	75-313
-lung	8	129	56-254	16	83	47-135	27	87	57-127
-melanoma	1	109	3-607	1	89	2-496	0	0	*******
-prostate	1	152	4-847	5	213	69-497	9	101	46-192
-testis	3	353	73-1032	0	0	*******	0	0	*******
-brain & other nervous system	5	258	84-602	3	105	22-307	5	233	76-544
-lymphatic leukaemia	0	0	*******	0	0	*******	4	364	99-932
-other malignant	4	426	116-1091	3	134	28-392	13	340	181-581
Circulatory System	40	97	69-132	126	113	94-135	207	91	79-104
-acute myocardial infarction	23	108	68-162	73	120	94-151	105	98	80-119
-arterio-sclerosis	2	145	18-254	5	128	42-299	17	148	86-237
-aortic aneurysm	1	143	4-797	5	242	79-565	13	239	127-409
Respiratory System	4	80	22-205	7	62	25-128	32	109	75-154
-chronic bronchitis, asthma & emphysema	3	175	36-511	4	76	21-195	19	133	80-208
Digestive System	4	59	16-151	19	135	82-256	21	134	83-205
-cirrhosis of the liver	4	100	27-256	17	189	110-303	5	78	25-182
-gallbladder diseases	0	0	*******	0	0	*******	5	472	153-1101

(shading included for reading ease only)

TABLE 4: Standardized Mortality Ratios by Years Since First Employment for Selected Causes, IDSP Mortality Study of Fire Fighters in Metropolitan Toronto (L'Abbé and Tomlinson, 1992)

	Years Since First Employment								
	< 20 Years			20-29 Years			≥ 30 Years		
Cause	Obs.	SMR	95% CI	Obs.	SMR	95% CI	Obs.	SMR	95% CI
All Causes	120	79	65- 94	143	95	80-112	514	99	91-108
All Cancers	28	120	77-170	36	94	66-131	135	106	89-125
-pharynx	0	0	*******	1	122	3-679	3	188	39-549
-rectum	1	125	3-696	2	146	18-527	10	179	86-329
-pancreas	1	100	3-557	2	95	12-344	11	157	78-281
-lung	1	23	1-128	13	103	55-176	40	100	71-136
-melanoma	1	91	2-507	1	130	3-724	0	0	*******
-prostate	0	0	*******	2	244	30-881	14	126	69-211
-testis	3	333	69-973	0	0	*******	0	0	*******
-brain & other nervous system	6	286	105-623	2	99	12-356	6	222	81-483
-lymphatic leukaemia	0	0	*******	0	0	*******	4	267	73-684
-other malignant neoplasms	1	143	4-797	4	288	78-737	15	306	171-505
Circulatory System	37	93	65-128	77	118	93-147	267	96	85-108
-acute myocardial infarction	22	102	64-154	48	126	93-167	135	102	86-121
-arteriosclerosis	1	100	3-557	3	165	34-482	20	141	86-218
-aortic aneurysm	0	0	*******	3	303	62-886	16	232	133-377
-diseases of veins, lymphatics, etc.	1	143	4-797	2	211	25-760	5	161	52-376
Chronic bronchitis, asthma & emphysema	0	0	*******	2	80	10-290	26	146	95-214
Cirrhosis of the liver	4	87	24-223	10	152	73-280	12	141	73-246
Gallbladder diseases	0	0	*******	0	0	*******	5	385	125-898

(shading included for reading ease only)

TABLE 5: Standardized Mortality Ratios by Age for Selected Causes, IDSP Mortality Study of Fire Fighters in Metropolitan Toronto (L'Abbé and Tomlinson, 1992)

	Age					
	< 60 Years			≥ 60 Years		
Cause	Obs.	SMR	95% CI	Obs.	SMR	95%CI
All Causes	337	91	81-101	440	97	88-107
All Cancers	87	107	86-132	112	104	86 125
pharynx	1	61	2-340	3	240	49-701
-rectum	4	138	38-353	9	191	87-363
-pancreas	4	97	26-248	10	170	81-313
-lung	22	91	57-138	32	97	66-137
-melanoma	2	93	11-336	0	0	*******
-prostate	2	153	19-553	14	130	71-218
-testis	3	268	55-783	0	0	*******
-brain & other nervous system	10	198	95-364	4	204	56-522
-lymphatic leukaemia	0	0	*******	4	336	92-860
-other malignant	9	304	139-577	11	266	133-476
Circulatory System	150	110	93-129	231	93	81-106
-acute myocardial infarction	93	117	94-146	112	100	82-120-
arteriosclerosis	4	113	31-289	20	149	91-230-
aortic aneurysm	3	163	33-473	16	245	140-398
-veins, lymphatics & other	3	148	31-433	5	181	59-422
Respiratory System	7	57	23-117	38	112	79-154
-chronic bronchitis, asthma & emphysema	2	43	5-155	26	155	101-227
Digestive System	20	96	58-148	24	156	100-232
-cirrhosis of the liver	18	130	77-205	8	138	60-272
-gallbladder diseases	0	0	*******	5	420	136-980

(shading included for reading ease only)

d) Cardiovascular disease among firefighters

- **Parameters of this Report**

An acute cardiac event such as a heart attack is adjudicated as a "chance event" or an "accident" by the WCB. Firefighters who have had heart attacks during the course of their employment have been compensated, albeit not always, for the period of disability resulting from the heart attack. The Ontario WCB, like most other Boards, does not usually compensate workers for periods of chronic disability resulting from cardiovascular disease.

The questions put to the Panel by the representatives of the firefighters concerned the relationship between cardiovascular *disease* and firefighting. While the Panel acknowledges that heart attacks or acute events are most often preceded by cardiovascular disease, the purpose of this Report is limited to a review of the data necessary to examine the relationship, if any, between cardiovascular disease and firefighting. The Panel will not be addressing specific compensation issues concerning acute events.

- **Medical terms**

It may be useful to define briefly the medical conditions which will be discussed below.

Atherosclerosis, which is a specific type of arteriosclerosis, results from the accumulation of cholesterol and other substances within the arteries. It has major effects on the coronary arteries, the cerebral arteries and the aorta, the main artery of the body. It causes a decrease in the coronary arteries' ability to allow blood flow to the heart muscle (coronary artery disease). Known risk factors for atherosclerosis are high blood pressure, high levels of cholesterol in the blood and cigarette smoking [158]. It may also produce narrowing of large arteries such as the aorta and its immediate branches, or weakening of the aortic wall leading to **aortic aneurysm.**

Ischemic heart disease results when the supply of oxygen to the heart muscle is inadequate to meet the demand. If ischemia becomes severe, a **myocardial infarction** or "heart attack" can occur. A myocardial infarction causes pathologic death of an area of heart tissue. Ischemic heart disease is common in the general population. In addition to atherosclerosis, ischemia may be caused by other factors that reduce oxygen delivery, such as exposure to carbon monoxide (CO), or that raise oxygen requirements, such as the production of high levels of adrenalin due to emotional and physical stress and strenuous activity [6].

- **Consultants' comments**

Three medical experts were asked to comment on the cardiovascular findings in the IDSP study. Each had a different interpretation of those findings.

Dr. J. K. Wilson wrote:

> "The latest statistics on coronary disease and myocardial infarction suggest only a very minimal increased risk (among firefighters) ... If a fire fighter does develop critical coronary artery disease then the acute stresses to which he is exposed in the course of his duties could certainly be an aggravating factor and could precipitate angina and it is possible that a very acute stress that he might be exposed to could result in rupture of an atherosclerotic plaque and set in motion the chain of events that leads to thrombus formation in a coronary artery and a resulting myocardial infarction that could be fatal.
>
> . . . .
>
> The information you have provided would suggest that there is a slight increase in susceptibility to arteriosclerosis in fire fighters. Whether a particular acute situation can be related to the actual firefighting duty or not would have to be assessed on an individual basis." [208]

Dr. C. D. Morgan offered the following opinion:

> "That physical and/or mental stress may precipitate myocardial infarction in individuals with pre-existing coronary heart disease is generally accepted but difficult to prove. The occupational environment of firefighting i.e. heavy physical exertion, heat, carbon monoxide, mental stress would appear sufficient to increase the risk of myocardial infarction in a predisposed individual. Even so, establishing cause and effect is difficult and the quality of evidence supporting an increased risk of myocardial infarction amongst firefighters is anecdotal. It remains less clear, and in my opinion unlikely, that the occupational environment of firefighting leads to an increased risk of remote myocardial infarction.
>
> . . . .

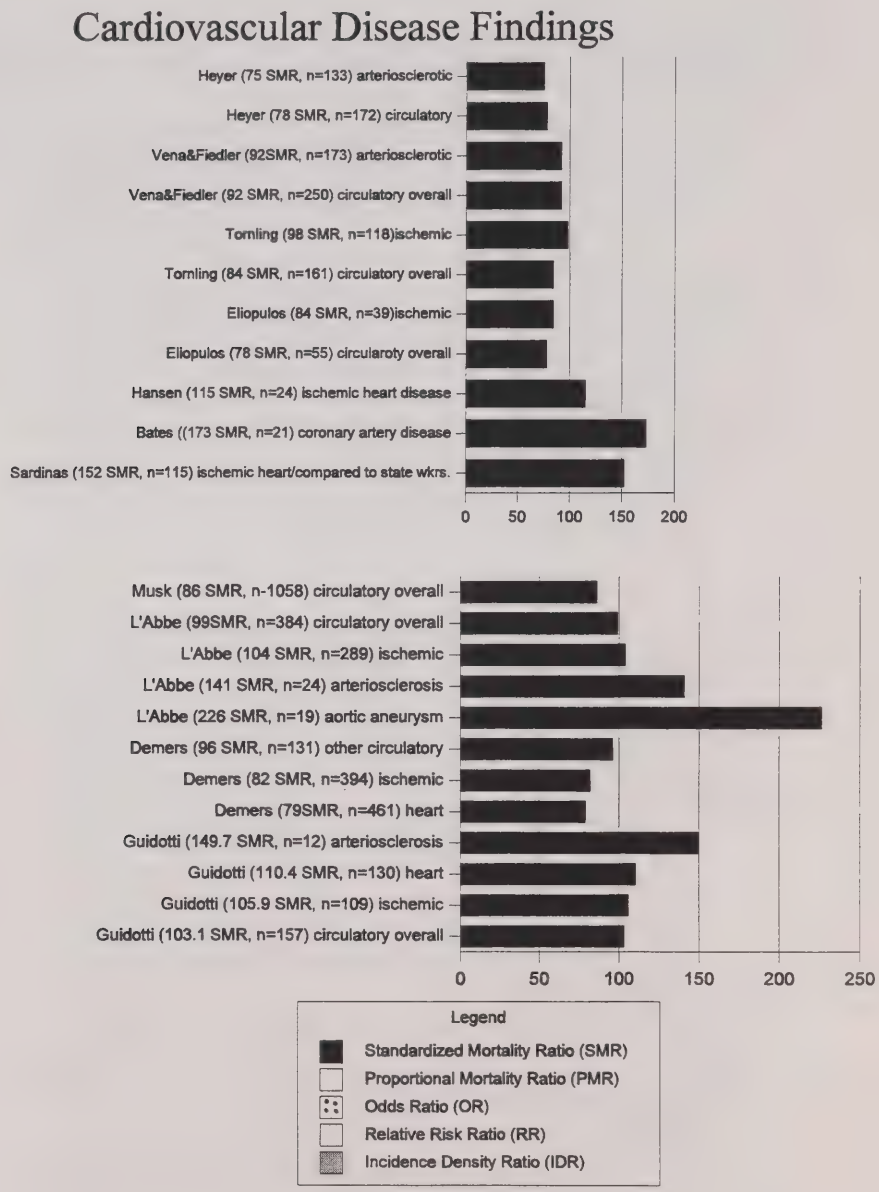
> In summary, despite many studies, there is insufficient evidence to support the contention that the occupation of firefighting is associated with an increased risk of cardiovascular disease. Indeed, the bulk of evidence argues against such a conclusion." [126]

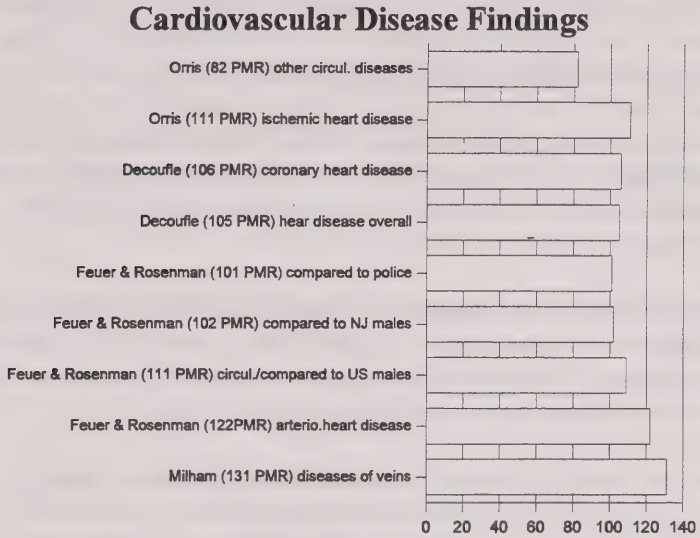
Dr. J. M. Melius, in contrast, thought that the findings should be interpreted as supporting an increased risk of cardiovascular disease among firefighters:

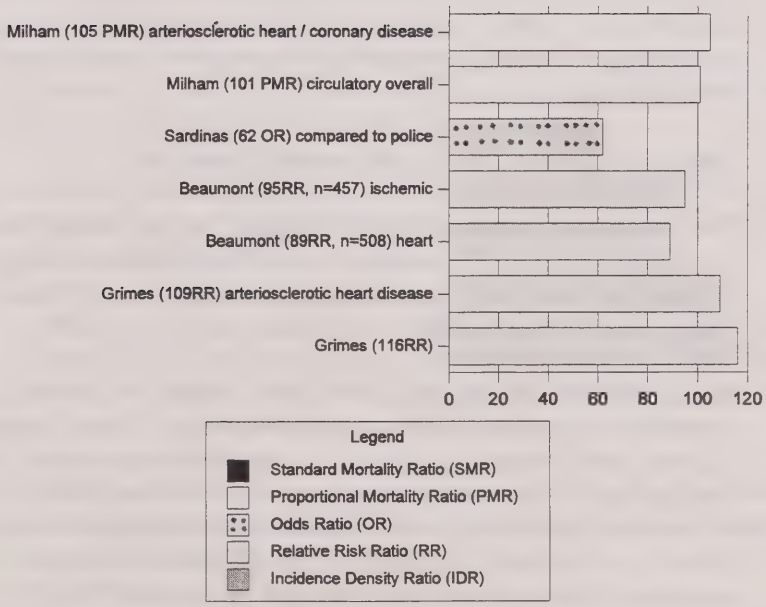
> "It is very difficult to draw conclusions about conditions such as cardiovascular disease (which is likely to be affected by employment selection) when comparing an occupational group to the general population.
>
> For example, [in the IDSP study] the mortality from myocardial infarction is slightly higher than the comparison population [SMR 107]. As stated in the report, this is higher than expected for a selected healthy population. One would expect a lower incidence, but this difference cannot be quantified in the absence of a control occupational group or other study approach. Hence, this finding gets overlooked in the discussion of the results.
>
> . . . .
>
> ... a number of epidemiological studies have found an increased mortality from cardiovascular disease despite the limitations from the healthy worker effect which would cause an underestimation of this risk ... This increased risk is supported by a number of other factors. First, fire smoke contains high levels of carbon monoxide and, on occasion, hydrogen cyanide. Both of these metabolic poisons increase the risk of acute cardiac events (amply supported by the carbon monoxide literature.) Carbon monoxide may also increase the risk of developing atherosclerosis (or accelerate its development) leading to a longer term risk of developing cardiovascular disease. These exposures cannot be completely avoided due to the limitation of current protective equipment (limited oxygen supply, etc.). Secondly, there are a number of other compounds in fire smoke which effect the respiratory system (particulates, acrolein, etc.). Fire fighters following a fire have a high prevalence of airway obstruction which can persist for some time after the fire. This diminished respiratory function also increases the risk of an acute cardiovascular episode. Thirdly, there are great physical stresses associated with fire fighting. Fire fighters work at very high levels of cardiac output for prolonged periods in a difficult environment which also places them at greater risk for an acute cardiovascular illness. Finally,

the mental stress of shift work and other fire fighting activities (rescue, etc.) may also increase the risk of cardiovascular disease." [119]

Fig. 3


Cardiovascular Disease Findings
Heyer (75 SMR, n=133) arteriosclerotic
Heyer (78 SMR, n=172) circulatory
Vena&Fiedler (92SMR, n=173) arteriosclerotic
Vena&Fiedler (92 SMR, n=250) circulatory overall
Tornling (98 SMR, n=118)ischemic
Tornling (84 SMR, n=161) circulatory overall
Eliopulos (84 SMR, n=39)ischemic
Eliopulos (78 SMR, n=55) circularoty overall
Hansen (115 SMR, n=24) ischemic heart disease
Bates ((173 SMR, n=21) coronary artery disease
Sardinas (152 SMR, n=115) ischemic heart/compared to state wkrs.
0 50 100 150 200
Musk (86 SMR, n-1058) circulatory overall
L'Abbe (99SMR, n=384) circulatory overall
L'Abbe (104 SMR, n=289) ischemic
L'Abbe (141 SMR, n=24) arteriosclerosis
L'Abbe (226 SMR, n=19) aortic aneurysm
Demers (96 SMR, n=131) other circulatory
Demers (82 SMR, n=394) ischemic
Demers (79SMR, n=461) heart
Guidotti (149.7 SMR, n=12) arteriosclerosis
Guidotti (110.4 SMR, n=130) heart
Guidotti (105.9 SMR, n=109) ischemic
Guidotti (103.1 SMR, n=157) circulatory overall
0 50 100 150 200 250
Legend
Standardized Mortality Ratio (SMR)
Proportional Mortality Ratio (PMR)
Odds Ratio (OR)
Relative Risk Ratio (RR)
Incidence Density Ratio (IDR)


Cardiovascular Disease Findings
Orris (82 PMR) other circul. diseases
Orris (111 PMR) ischemic heart disease
Decoufle (106 PMR) coronary heart disease
Decoufle (105 PMR) hear disease overall
Feuer & Rosenman (101 PMR) compared to police
Feuer & Rosenman (102 PMR) compared to NJ males
Feuer & Rosenman (111 PMR) circul./compared to US males
Feuer & Rosenman (122PMR) arterio.heart disease
Milham (131 PMR) diseases of veins
0 20 40 60 80 100 120 140


Milham (105 PMR) arteriosclerotic heart / coronary disease
Milham (101 PMR) circulatory overall
Sardinas (62 OR) compared to police
Beaumont (95RR, n=457) ischemic
Beaumont (89RR, n=508) heart
Grimes (109RR) arteriosclerotic heart disease
Grimes (116RR)
0 20 40 60 80 100 120
Legend
Standard Mortality Ratio (SMR)
Proportional Mortality Ratio (PMR)
Odds Ratio (OR)
Relative Risk Ratio (RR)
Incidence Density Ratio (IDR)

- **Studies of cardiovascular disease among firefighters**

Findings from the various studies are illustrated in Fig. 3. More detail is provided in Table 10 which appears at the end of this chapter.

The IDSP study (L'Abbé and Tomlinson, 1992)

The Panel has chosen to highlight the findings of the study of Toronto firefighters. As with any epidemiological study, however, the findings of an individual study are more compelling when they are confirmed by other research.

As shown in Table 2, deaths from ischemic heart disease and myocardial infarction were only slightly higher than the rates for the general Ontario male population. Deaths attributed to arteriosclerosis were elevated but did not reach statistical significance.

A statistically significant increase did occur, however, in deaths among Toronto firefighters caused by aortic aneurysms. Sixteen of those 19 cases of aortic aneurysm occurred in men over 60 years of age.

The SMR for all causes was lower than expected. The authors initially expected that pre-employment screening would have led to a lower rate of death from cardiovascular disease compared to other causes of death among firefighters (the healthy worker effect); however, the risk of death from cardiovascular disease was about 10% *higher* than the risk of death from all other causes [193].

Other firefighter mortality and morbidity studies

The Panel found the analysis of the data about cardiovascular disease difficult because the authors of the studies described their findings using different disease classifications. This resulted in over forty statistical analyses for different cardiovascular disease outcomes.

In considering this complexity, the Panel attempted to determine if it was reasonable to rely on the distinction between arteriosclerotic disease and ischemic heart disease as the primary cause of death as recorded on death certificates. There is not a wealth of literature to assist the Panel; however, two articles that discuss the accuracy of death certificate data noted that the reliability of the identified primary cause of death decreases in the case of concurrent chronic disease among an aging population [172, 99]. In a paper which specifically referred to the accuracy of cardiovascular disease on death certificates, the authors noted that cerebral stroke was properly classified in 84% of the cases and cardiac disease in 66% of the cases [65].

The Panel also considered the pathological or disease process for the two most commonly used classifications, arteriosclerosis and ischemic heart disease. Because arteriosclerosis leads to ischemic heart disease, the Panel decided that it would be reasonable to group the data for each of these outcomes into one analysis. This grouping of data leads to more compelling evidence because it helps to overcome problems with small numbers of cases in each category. Furthermore, inconsistencies of classifications based on the judgement of individual physicians is overcome by grouping like categories together.

Accordingly, the Panel has structured its analysis in the following way:

- cardiovascular disease generally;
- arteriosclerosis and ischemic heart disease combined; and
- aortic aneurysms.

- **Cardiovascular disease generally**

In 1959, Mastromatteo reported a statistically significant elevation in the rate of "cardiovascular-renal" deaths among Toronto firefighters between 1921 and 1953 [109]. Despite the care taken, the author of this very early and important study was able to achieve a follow-up of only about 78.4% of the total cohort. For this reason, its results must be viewed with some caution.

Grimes and colleagues (1991) noted a statistically significant elevation in the risk ratio (RR 1.16; 95% CI 1.10-1.32) for all circulatory diseases in a small group of 205 Hawaiian firefighters [56].

Three firefighter studies found increases in cardiovascular mortality which were not statistically significant, as reported by Hansen (1990), Milham (1983) and Guidotti (1993).

A statistically significant decrease in circulatory disease mortality in a large cohort of Boston firefighters was reported by Musk et al. (1978). Similarly, Tornling and co-workers (1994) found a statistically significant decrease in circulatory disease deaths among 1116 Swedish firefighters.

The authors of five other studies conducted in Canada, the US, Denmark and Australia either reported fewer or about the expected number of deaths from circulatory diseases [32, 43, 10, 68, 202].

Another mortality study, by Decoufle et al. (1977), combined data from available records about firefighters and stationary engineers. The authors reported increases in heart disease deaths, particularly among those under 55

years of age. Because of the combined cohort, the results for firefighters are less than ideal.

In addition to the mortality data, there are data about the cardiac health of living firefighters. As part of the US *Normative Aging Study*, 171 firefighters were followed for ten years. The authors concluded that firefighters had no significant differences in their baseline risk factors compared with non-firefighters, and they had no statistically significant difference in the incidence of coronary heart disease [34].

In another setting, Barnard and colleagues (1975) investigated the prevalence of ischemic heart disease among Los Angeles firefighters. They conducted medical examinations and near-maximal stress testing using treadmills on 90 randomly selected firefighters and compared them with 232 insurance underwriters from the same city and in the same 40-59 year age range. The risk factors for both groups are shown in Fig. 4 below.

Fig. 4. Risk factors for ischemic heart disease among Los Angeles firefighters compared to underwriters, 1975

	90 firefighters	232 underwriters
▸ cholesterol over 260 mg% (6.72 mmol/L):	12%	18%
▸ blood pressure 160/90 mm Hg & over:	2%	25%
▸ current smokers:	32%	26%

Despite lower risk factors (other than smoking), ten percent of the firefighters had ischemic stress test results, compared with only eight percent of the underwriters. The authors found this incidence of ischemic stress tests surprising, since firefighters are a medically-selected group, and suggested that ischemic heart disease may be caused by emotional stress, heat stress and inhalation of pollutants on the job [7].

The cardiovascular fitness of firefighters in Montreal was compared with the general Canadian population recently by Horowitz and Montgomery (1993). They found that firefighters had higher flexibility and muscular endurance, but had lower cardiovascular endurance and were considerably heavier. The lower cardiovascular endurance was attributed to elevated body mass and an elevated heart rate response to exercise. The authors speculated that the heart

rate response may also reflect long-term effects of exposure to carbon monoxide and other chemicals. This decline in cardiovascular health was also noted in previous studies of firefighters.

- **Arteriosclerosis/Ischemic heart disease**

Two studies of firefighters' mortality found statistically significant increases in ischemic heart disease. One of these is the largest firefighter study, conducted by Orris et al. (1992), involving 3084 deaths among Chicago firefighters. The authors reported a Proportionate Mortality Ratio (PMR) for ischemic heart disease of 111 (95% CI 106-118).

Sardinas and colleagues (1986) reported an SMR of 1.52 (95% CI 1.23-1.81) for ischemic heart disease among 306 Connecticut firefighter deaths compared to other male workers in that state.

Between 1970 and 1983, Bates (1987) noted an SMR of 1.73 (95% CI 1.12-2.66; $p<0.005$[8]) for coronary artery disease (arteriosclerosis) among Toronto firefighters. The risk was higher for those who were aged 45-49 in the 1970's. One of the reasons that the author suggested for the decline in the SMRs was that in the late 1970's the Toronto Fire Department made a policy change to allow firefighters who showed evidence of coronary artery disease to retire or be transferred to non-firefighting situations. As a result, the post-1970 group of firefighters was a lower risk population.

Feuer and Rosenman (1986) also identified a statistically significant increase in arteriosclerosis among 263 New Jersey firefighter deaths, with a PMR of 1.22 ($p<0.05$) compared to US males. The PMR was lower (1.11) compared to state males, and lower still (1.06) when compared to police officers, suggesting that the difference was smaller when more appropriate comparison groups were used. Other cardiovascular deaths were near or below expected rates.

As explained above, the Panel combined the findings of the various studies for arteriosclerosis and ischemic heart disease. The results are captured in Table 6.

[8] A "p-value" refers to the probability that chance produced an apparent excess of disease. For example, "$p<0.05$" means there is a less than a 5% likelihood that the observed result occurred by chance.

Table 6: Arteriosclerosis/ischemic heart disease combined SMRs

Authors	SMR	Authors	SMR
Sardinas	152 *	Tornling	98
Guidotti	150	Beaumont	95
L'Abbé/Tomlinson	141	Vena/Fiedler	92
Feuer/Rosenman	122 (PMR)*	Demers	82 *
Hansen	115	Eliopulos	84 *
Orris	111 *	Heyer	75
Grimes	109	Milham	105

* indicates a statistically significant increase or decrease

In assessing the statistical data about cardiovascular mortality among firefighters, the majority of the reviewers commented on possible bias that would have been introduced by the healthy worker effect. There seems to be no doubt that when firefighters enter the service they have good cardiovascular health. Whether that level of health continues throughout their employment is debatable as evidenced by the studies of Los Angeles and Montreal firefighters. What is no longer debatable is that the rates of cardiovascular illness are strongly influenced by the healthy worker effect. Since firefighters are selected for their excellent health, moderate increases in cardiovascular disease compared to the general population are probably underestimates of the magnitude of the true hazards of firefighting.

The authors of the IDSP study articulated this problem in the following way.

> "Fire fighting is unlike most occupations in that to become a fire fighter, one must pass a medical examination, an aptitude test and tests of physical skill. The six fire departments in this study all had approximately the same entrance requirements. Although the requirements were not so stringent in the past, to be hired, one has always had to be physically fit. It does not seem unreasonable to assume that at least at the start of his career, the fire fighter is much healthier than the average person in the general population. When the physical nature of fire fighting work is considered, it becomes apparent that to continue being a fire fighter demands a level of health higher than that of the general population." [86]

The Panel consulted Dr. B. C. K. Choi, of the University of Toronto, about various ways to reduce the impact of the healthy worker effect, particularly on the cardiovascular and respiratory findings [25]. Dr. Choi's reports

discussed several approaches and recommended three options in order of preference:

1. The most highly recommended option was to compare firefighters with other firefighters within the cohort by length of employment. (As Dr. Choi noted, many of those analyses had already been conducted by L'Abbé and Tomlinson);

2. The second most highly recommended approach was to choose another working cohort such as Ontario Hydro forestry workers; however, there were too few deaths among the Ontario Hydro cohort to allow for a reliable comparison;

3. The final option recommended was to compare the Toronto firefighters with police officers from the Demers et al. (1992) study entitled *Mortality among firefighters from three northwestern United States cities*.

The Panel attempted to identify another working cohort but found no other cohort which appeared to be suitable for comparison with firefighters.

The Panel did, however, conduct additional calculations in an attempt to overcome the healthy worker effect. The combined arteriosclerosis and ischemic heart disease rates were compared with the rate of mortality from all causes in each study, if the necessary information was available. This is a generally accepted method of attempting to correct for the potential bias of the healthy worker effect [75, 185]. The ratio of the SMR for arteriosclerosis and ischemic heart disease to the SMR for all causes could be calculated for nine studies[9], as shown in Table 7. As can be seen, the rate of cardiovascular disease was higher than the overall SMR in seven of the nine studies.

[9] PMRs, ORs and RRs were not included in this analysis because they are measurements which are not comparable with SMRs.

Table 7: Combined arteriosclerosis/ischemic heart disease, compared to all causes of death

Authors	Arterio./Ischemic SMR	All Causes SMR	Arterio./Isch.SMR / All Causes SMR X 100
Guidotti	150	96	156
L'Abbé/Tomlinson	141	94	150
Hansen	115	99	116
Tornling	98	82	120
Beaumont	95	90	106
Vena/Fiedler	92	95	97
Eliopulos	84	80	105
Demers	82	81	101
Heyer	75	76	99

Average: (Arteriosclerosis & Ischemic SMR/All Causes SMR) = 117

Discussion

While the Panel has the benefit of a number of mortality studies of firefighters, it must also deal with the inconsistent findings of those studies with respect to cardiovascular disease.

Of the fifteen mortality studies that examined cardiovascular illness, nine found rates in excess of the comparison groups. Four of the studies reported excesses which were statistically significant. The SMRs ranged from 173 to 105. The remaining six studies reported fewer deaths from cardiovascular causes than expected; two of the results were statistically significant. The SMRs ranged from 98 to 75.

The weight of the evidence suggests that there may be a relationship between cardiovascular disease and firefighting. The strength of the association, however, is not very powerful. Before correcting for the healthy worker effect the average SMR was 104, and 117 after the correction.

It is important to note that the study with the largest number of firefighters, Orris et al. (1992), did find statistically significant excesses. In addition, when Bates compared Toronto firefighters to Toronto males, the SMR for coronary heart disease was 173 and statistically significant [8]. Finally, the study conducted by Hansen (1990) used another working population, Danish civil servants, as the comparison group and found a statistically significant SMR of 152.

FIG. 5: CIRCULATORY DISEASE MORTALITY AMONG SEATTLE FIREFIGHTERS, 1945-1983, BY DURATION OF EMPLOYMENT:

Years worked	SMR	(95%CI)	n
<15	63	(39-96)	21
15-29	75	(62-91)	107
30+	103	(75-138)	44

n=number of deaths
(Heyer et al., 1990)

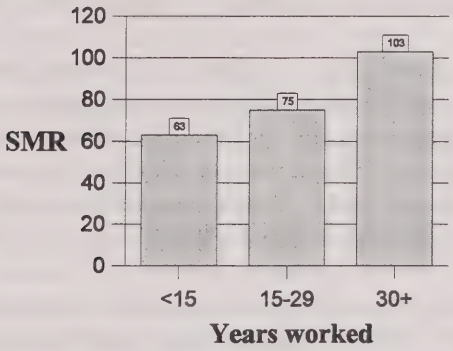
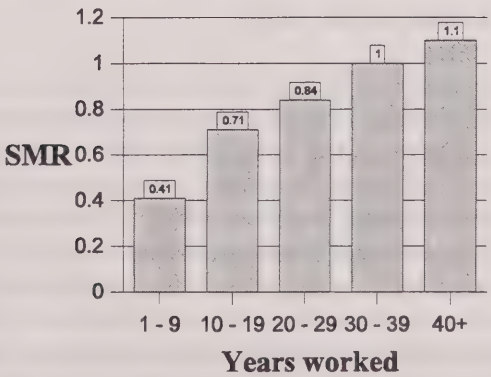


FIG. 6:CIRCULATORY DISEASE MORTALITY AMONG BUFFALO FIREFIGHTERS, 1950-1979, BY DURATION OF EMPLOYMENT:

Years worked	SMR	(95%CI)	n
1-9	0.41*	(0.146-0.795)	6
10-19	0.71	(0.422-1.082)	18
20-29	0.84	(0.639-1.058)	61
30-39	1.00	(0.823-1.206)	106
40+	1.10	(0.839-1.402)	59
total	0.92	(0.809-1.037)	250

*p<0.05 n=number of deaths
(Vena & Fiedler., 1987)



- **Dose-response analyses**

The existence of a dose-response relationship is usually a clear indicator of a relationship between exposure and disease.

With respect to cardiovascular disease and firefighting, dose-response relationships were not consistently identified. Eleven studies conducted dose-response analyses; in nine of them no such pattern emerged [47, 34, 10, 56, 63, 43, 32, 195, 59]. Even more importantly, two of these nine studies used indices of actual fire exposure, rather than only duration of employment, and they failed to identify a dose- response relationship [59, 195].

As with any pattern, however, there are variations. All subgroups of Heyer and colleagues' (1990) Seattle cohort had the expected rate of mortality due to circulatory diseases, except for firefighters with 30 or more years of exposure. An overall dose-response trend was shown by the steadily increasing SMRs for circulatory disease based upon duration of exposure (Fig. 5). A similar increasing trend occurred in the analysis based upon time since first exposure.

A clear dose-response trend also appeared in Vena and Fiedler's (1987) Buffalo cohort for all circulatory diseases (Fig. 6) and for arteriosclerosis, both of which also increased fairly consistently with increasing latency; however, the SMR for the group with the lowest exposure was only 110.

The Panel must address the significance and possible explanations for the absence of consistent patterns of dose-response relationships.

On this point, Sir Austin Bradford Hill, in his work *The environment and disease: Association or causation?*, wrote:

> "Often the difficulty is to secure some satisfactory quantitative measure of the environment which will permit us to explore this dose-response." [69]

It is particularly difficult to identify quantitative measures of exposures in the case of firefighters who are neither consistently exposed to the same substances nor exposed for regular periods of time. Consistently, industrial hygienists and toxicologists cautioned the Panel against rigid reliance on their evaluation of the contaminants in fires. There are few studies that attempt to quantify the contents of fire smoke and even fewer studies that investigate all of the varied kinds of contaminants at fire sites. In fact, the vast majority of studies do not even attempt to quantify number of fires attended but rely strictly on duration of employment as a surrogate for exposure. The Panel was unsuccessful in its attempt to gather information about average or specific numbers of fires for stations in Ontario. As a result, it is impossible to know the "dose" experienced by firefighters. Without that information, a dose-response trend cannot be investigated.

The dose-response relationship may also be obscured by a survivor effect. A survivor effect is particularly likely in firefighters, since cardiovascular disease would in most instances lead the firefighter to change employment. Therefore, the firefighters with the longest duration of employment would be heavily loaded with survivors, that is, with those who had not contracted cardiovascular disease.

A method for investigating dose-response in the presence of a strong survivor effect has been proposed by Fox and Collier (1976). This method examines dose-response in terminated workers who have had at least 15 years of employment (to minimize the healthy worker effect). Dr. K.A. L'Abbé re-analyzed the data from the IDSP study of Toronto firefighters using this method. Among the terminated workers a dose-response trend by duration of employment was not found. The SMR was lower after 25 years duration than at 15-19 or 20-24 years duration [87, 88].

Another explanation for the absence of a dose-response pattern was proposed by Feuer and Rosenman (1986). They found an inverse relationship between mortality from arteriosclerotic heart disease and time since first employment which suggested to them that the firefighters who were most susceptible to heart disease were affected early in their careers. This interpretation introduces the concept of an innate susceptibility to a disease being aggravated by physical and psychological occupational factors.

Discussion

Nine of the eighteen firefighter studies provided dose-response analyses of the relationship between firefighting and cardiovascular disease; however, a dose-response trend was identified in only two of them. The lack of a dose-response trend does not rule out an association, however, because it is impossible to measure the "dose" experienced by firefighters and because of variations in individual susceptibility in the development of cardiovascular disease.

- **Potential causative agents**

Carbon monoxide

Authors McDiarmid et al. (1991), Jankovic (1991) and Nadon et al. (1992) have all independently identified carbon monoxide as probably the most common exposure experienced by firefighters.

Although controversy exists over the long-term effects of repeated exposure to relatively low concentrations of carbon monoxide, it is generally agreed that CO-induced hypoxemia produces the same pathology as oxygen deprivation. Permanent tissue damage would result only from acute exposures leading to tissue hypoxia and this damage is believed to be cumulative (that is, repeated

acute exposures causing repeated tissue hypoxia leads to increases in permanent tissue damage) [188]. Such acute exposure is likely the type of exposure experienced by firefighters.

As identified by Dr. Morgan [126] and Dr. Melius [119], there is also experimental evidence that carbon monoxide increases the risk of cardiovascular disease.

Authors Guidotti and Clough (1992) have proposed two possible reasons why an increase in heart disease might occur among firefighters: sudden stress and carbon monoxide exposure, which has also been linked to angina.

Other authors have concluded that regular, severe carbon monoxide exposure increases the risk of ischemic heart disease [173] and raises the incidence of ventricular arrhythmias during exercise in patients with coronary artery disease [174].

Intermittent and intense exposure to physical and emotional stress

It is generally accepted that the demands on the cardiovascular system increase significantly in response to physical or emotional stress. For example, there is evidence that firefighters' heart rates dramatically increase when responding to an alarm. Studies have shown that the increase averages 50 beats within 30 seconds of the alarm, which persists until arrival at the fire, a much greater elevation than required by the physical exertion [60, 6]. These extra demands on the cardiovascular system may cause angina or a more serious result such as a heart attack.

What is less clear is the effect of chronic stress or intermittent bouts of extreme stress. While popular thought and literature has often directly related heart disease to stress, the scientific data does not support this proposition.

The National Heart Foundation of Australia reported:

> "The available research evidence provides little scientific grounds for concluding in individual cases that the continuing chronic stress of any particular job may have contributed directly to coronary heart disease; or recommending that any individual should avoid certain jobs or change to another job to prevent coronary heart disease." [161]

The authors noted, however, that there might be an indirect relationship between heart disease and exposure to stressful activities. Specifically, they wrote:

> "From the large number of studies that have been carried out in this area, it is plausible that stress may promote or aggravate the established coronary risk factors and thus pre-dispose persons indirectly to ischaemic heart disease. However, the extent of this effect is uncertain." [161]

Discussion

The most significant and important exposure with respect to cardiovascular disease faced by firefighters is carbon monoxide. Although there is some disagreement about the weight of the current evidence, there is no dispute that there is a growing body of experimental evidence to support the connection between the disease and carbon monoxide exposure. There is also no dispute that firefighters are exposed to carbon monoxide.

The evidence about the other identified potentially harmful exposures is not as conclusive. There is, however, some support that chronic stress will aggravate and accelerate pre-existing heart disease.

Summary of the evidence regarding cardiovascular disease, arteriosclerosis and ischemic heart disease

The results of studies that have surveyed the existence of heart disease among firefighters are not conclusive; nonetheless, the Panel has been able to identify the following trends.

- The majority of reports show an excess of cardiovascular disease among firefighters.
- The largest available study of firefighters, which surveyed the causes of 3084 firefighter deaths [147], demonstrated a statistically significant excess of deaths that had occurred due to ischemic heart disease.
- An attempt was made to control for the healthy worker effect by computing the ratio of the SMR for heart disease to the SMR for all causes of death. The ratio was 117, consistent with a modestly increased effect.
- This excess is not consistently found among all studies, nor is it a very large excess, which weakens the strength of the association between firefighting and cardiovascular disease.

- Because of the influence of the survivor effect and a susceptibility factor, the absence of a dose-response does not negate the possibility of an association between cardiovascular disease and firefighting.

- There is an important known exposure, carbon monoxide, which is present at all fires, that provides some biological explanation for the finding of an excess incidence of cardiovascular illness among firefighters.

- **The Panel's conclusions and finding regarding cardiovascular disease, arteriosclerosis and ischemic heart disease**

These data led the Panel to conclude that there is evidence, although not consistent evidence, of an association between firefighting and heart disease. In those powerful studies which identify an excess, the association is statistically significant. This excess can be explained by the exposures experienced by firefighters. While there was not unanimous agreement among the IDSP's expert cardiovascular consultants about the potential link between firefighting and cardiovascular disease, those consultants' opinions were solicited before the Panel had conducted the additional investigations and analyses described above. As a consequence, they were not able to consider all the evidence that was before the Panel.

In summary, the evidence which supports an association is greater than the evidence which refutes an association. Accordingly, the Panel makes the following finding.

- **A probable connection exists between cardiovascular disease and the occupation of firefighting.**

The Panel reviewed the criteria used in the past for deciding whether a disease should be added to one of the Schedules, then applied the same principles to the evidence about cardiovascular disease among firefighters. Although there are some impressive findings of excess cardiovascular disease, they are not consistently found. Furthermore, the majority of studies surveyed which report an excess do not establish a statistically significant excess. Finally, the identified contributors to heart disease, carbon monoxide and chronic stress, have been experimentally but not conclusively identified as causes of chronic heart disease. This body of evidence has caused the Panel not to recommend that cardiovascular disease be added to either Schedule 3 or 4. The Panel does recommend, however, that firefighting be recognized as a risk factor in the development of cardiovascular disease.

The Panel's recommendation regarding cardiovascular disease, arteriosclerosis and ischemic heart disease

- **When the Board adjudicates a claim for heart disease from a firefighter, the occupation of firefighting should be recognized as a risk factor for cardiovascular disease and should be weighed with the other risk factors such as hypertension, smoking and family history when determining entitlement under the *Workers' Compensation Act*.**

e) Aortic aneurysms

- **Mortality due to aortic aneurysms**

The IDSP study found a statistically significant increase in the risk of death from aortic aneurysm among Toronto firefighters, with an SMR of 226 (95% CI 136-353).

In his comments on these data, Dr. C. Morgan suggested that the statistically significant increase in aortic aneurysm mortality may have resulted from multiple tests of significance, particularly in the absence of a prior hypothesis. This refers to the possibility that when numerous disease outcomes are examined, statistically significant associations will be found for some of them by chance alone.

The Panel asked one of the study's authors, George Tomlinson, to investigate this potential source of bias. He used the method proposed by Schweder and Spjotvoll (1982) in which a simultaneous evaluation of all the tests is made on a plot of cumulative p-values using the observed significance probabilities. The points corresponding to a *true* null hypothesis[10] should form a straight line, while those for a *false* null hypothesis should deviate from this line.

There were 92 disease outcomes, excluding those with fewer than two deaths expected. By the Schweder and Spjotvoll method, 12 of the 92 were considered to be false null hypotheses. Among the 12, aortic aneurysm had a notably small p-value of 0.001, hence it is probably a real association rather than being the result of multiple tests of significance [193].

Demers et al. (1992) studied the mortality of firefighters in the northwestern United States. They identified a statistically significant increase in the rate of death from "diseases of the arteries, veins and pulmonary circulation" (which includes aortic aneurysms) among firefighters with 30 or more years of employment (SMR 1.99 [1.3-2.9]) compared to United States white men[11]. The rate was also increased when firefighters were compared to police officers. Like the IDSP findings for Toronto firefighters, neither police nor firefighters from the northwestern US experienced an overall increase in cardiovascular mortality.

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10 The "null hypothesis" states that there is no difference between the groups being compared.

11 Mortality among firefighters from three northwestern U.S. cities, 1945-89:

"Diseases of the arteries, veins, and pulmonary circulation" among firefighters with 30 or more years of employment: SMR 1.99; 95% CI 1.3-2.9; 25 deaths. [32]

In order to learn more about the incidence of aortic aneurysms in other groups of firefighters, the Panel asked Dr. Demers to re-analyze the northwestern US firefighter data and provide the Panel with a mortality analysis for aortic aneurysms alone. The results are shown in Table 8.

Table 8: Mortality due to **Aortic Aneurysms** Among Northwestern US Firefighters and Police **compared to US National Rates for 1950-1989**

	Follow-Up: 1950-1989 Obs/Exp SMR (95% CI)	Follow-Up: 1970-1989 Obs/Exp SMR (95% CI)
All Firefighters	12/12.1 0.99 (0.51-1.73)	5/8.73 0.57 (0.18-1.33)
30+ yrs. employed	9/3.64 **2.47 (1.13-4.69)**	4/2.14 1.87 (0.48-4.21)
30+ yrs. after hire	11/10.5 1.05 (0.52-1.88)	5/7.77 0.64 (0.21-1.49)
All Police	0/6.38 1.57 (0.75-2.89)	6/4.89 1.23 (0.45-2.68)

Dr. Demers commented as follows:

> "The analyses that were performed indicate that long-term Seattle, Portland, and Tacoma firefighters may have a higher risk of death due to aortic aneurysms than the general population. However, due to the limitations which I will discuss below, I would only consider the SMRs in [Table 8] to be a rough estimate of the actual risk of aortic aneurysms.
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> . . . .
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> United States national white male rates for death due to aortic aneurysms between 1979 and 1990 were used because they were the only rates available and they may not be completely valid for a number of reasons. Disease-specific death rates have generally decreased over time due to improvements in health care and living conditions. However, some rates may increase as an artifact of changes or improvements of diagnostic procedures or coding practices. I am not sure in what direction, or by how much, the death rates for aortic aneurysms may have changed, but I have attached the US national rates for all deaths due to diseases of the arteries, veins and pulmonary circulation as an example. In this broad category of disease, the only big changes over time were among those 75 or older and it is important to remember that in the early decades of follow-up very few

> firefighters fell into this age category. Mortality rates can also vary by region and I suspect that, at least in recent years, the Pacific Northwest may have had lower rates. For example, the death rates per 100,000 population for diseases of the heart ... in 1987 for Washington and Oregon states were 255 and 295, respectively, while the national rate was 312. Unfortunately, I do not have more detailed information.
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> . . . .
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> One thing that is certain is that, due to the selection criteria for becoming a firefighter and the health needed to remain a firefighter for 30 or more years, one should expect that long-term firefighters would have a lower risk, and not higher, than the general population. There are some additional limitations that should be borne in mind. All of these comparisons are based on a relatively small number of deaths among both the firefighters and police. I am not a believer in the strict `<0.05' interpretation of statistical significance, but I think the width of the confidence limits gives you a good idea of the statistical precision of these estimates. It is also important to remember that death certificates are a poor source of information concerning aortic aneurysms." [33]

The Panel also asked Dr. Demers to conduct a comparison of the mortality from aortic aneurysms among Toronto firefighters with that among northwestern US firefighters and police using police as the comparison group in each case. His findings are shown in Table 9.

Table 9: Mortality due to **Aortic Aneurysms** Among Firefighters from Metropolitan Toronto and Northwestern US **compared to Police** (10 deaths) from Northwestern US

	Toronto Firefighters IDR [12] (95% CI)	Northwest US Firefighters IDR (95% CI)
Total Cohort	1.48 (0.67-3.29)	0.56 (0.23-1.34)
30+ yrs. employed	1.09 (0.46-2.58)	1.43 (0.48-4.21)
30+ yrs. after hire	1.26 (0.55-2.88)	0.71 (0.26-1.93)
	(20 deaths)	(12 deaths)

[12] "IDR" = Incidence Density Ratio, which means the ratio of age-matched incidence of disease in one group to the age-matched incidence in the comparison group.

Dr. Demers interpreted the findings in the following way.

> "Although the Metropolitan Toronto firefighters had a somewhat elevated risk (of aortic aneurysm) compared to the Northwest US police, the Northwest US firefighters did not. The results were also inconsistent when mortality among only firefighters employed for 30 or more years or at least 30 years after first employment was examined.
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> . . . .
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> There are some important limitations that should be kept in mind when interpreting the results of these analyses. First, the validity of the comparison of Toronto area firefighters to police from three Northwest US Cities is questionable. Although police may seem an ideal comparison group for firefighters, the stresses of police work, the lower physical fitness requirements necessary for police work (after hire), and the life style of police officers could potentially alter their risk of circulatory disease and make them a less than ideal comparison group. In addition, disease rates and smoking rates may differ between regions and the way that death certificates are completed and vital statistics compiled may also differ somewhat between the Northwest US and Ontario. I am not sure how important any of these factors might be, but I would be cautious in interpreting the results because of them.
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> . . . .
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> The analyses that were performed indicated that metropolitan Toronto firefighters may have a higher risk of death due to aortic aneurysms than the police comparison group but the numbers are too small to say this with any certainty. In addition, the relative risks among firefighters employed for at least 30 years or at least 30 years after first employment were less elevated. There was little evidence that Northwest US firefighters were at higher risk than the police from the same cities. The differences in relative risk observed between the metropolitan Toronto firefighters and Seattle, Portland, and Tacoma firefighters could be due to real differences in exposure or some of the other factors discussed above, such as chance." [33]

While aortic aneurysms do not appear to be increased in other firefighter mortality studies, an increase could have been obscured by the authors' use of broader categories of diseases such as "diseases of the arteries, veins and pulmonary circulation", as seen above. Because of the significance of findings in its study and the potential for confirming data from other studies, the Panel decided that this issue warranted further investigation. Accordingly, the authors of other firefighter studies were asked to re-analyze their data and investigate the incidence of aortic aneurysms specifically.

The largest study involved 3084 deaths between 1940 and 1988 among Chicago firefighters, reported by Orris et al. (1992). The updated data, which now includes 3314 deaths, were re-analyzed for the Panel by Dr. Paul Targonski. There was no overall increase identified for the period from 1948 to 1982. There was one finding of a statistically significant increase for the period from 1953 to 1957[13].[190]

Dr. J. Vena provided findings for aortic aneurysm for 1867 Buffalo firefighters from the 1987 study reported by Vena and Fiedler (1987). His analysis showed an overall SMR of 163 (95% CI 65-337). There were SMRs well above 200 in many subgroups[14]. A cluster of four deaths occurred in the group with 35-39 years since first exposure, resulting in an SMR of 547 (95% CI 149-1401) [203].

When Dr. T. L. Guidotti's 1993 data on 3328 Alberta firefighters were re-examined, he found that only two firefighters died of this cause during the study period covering 1927 to 1987. One was in the age group 55-59; the other, 70-74. There was no similarity between their cumulative or weighted exposures, but they both died in the period between 1985 and 1987 [58].

Dr. K. Rosenman was also asked to provide a further analysis of the original data from his 1986 report on New Jersey firefighter mortality [47]. Unfortunately, that was not possible because the original data are no longer available [163].

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[13] Mortality study of Chicago firefighters:

Overall crude PMR for aortic aneurysms during 1948-1982: 1.159; 95% CI 0.715-1.531;
Adjusted PMR for aortic aneurysms during 1953-1957: 3.115 ($p<0.05$); 95% CI 1.142-6.697. [190]

[14] Deaths from aortic aneurysm among City of Buffalo firefighters, 1950-1979:

Years worked	SMR	95% CI	Number
0-24	-	--	0
25-29	251	30-907	2
30-34	249	30-899	2
35-39	278	57-812	3 [203]

- **Consultants' comments**

The three experts consulted by the Panel had conflicting views about the possible role that occupation played in the increase in mortality due to aortic aneurysm found in the IDSP study.

Dr. J. Wilson stated:

> "This [increase in aortic aneurysms] comes as a surprise. As an explanation for this one has to consider the whole problem of arteriosclerosis as it affects the aorta and other arteries particularly the coronary arteries. There was only a minimal increase in mortality from ischemic heart disease and acute myocardial infarction and no increase in mortality from cerebral vascular disease. Why there should be an increased risk of aortic aneurysm in fire fighters is purely conjecture.
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> . . . .
>
> One can only ask the question as to whether sudden acute stresses both physical and mental can increase heart rate and this is well documented but it also increases blood pressure and during the acute stress the blood pressure may be significantly elevated and this could lead to more sheer stress on the endothelial cells of the aorta.
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> . . . .
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> Question 3 deals with the increased risk of aortic aneurysms in those over 60 years of age and often those who have terminated work. This is to be expected. It is quite unusual to have aortic aneurysm in younger people and the majority would be in the over 60 age range. It takes considerable time for sufficient arteriosclerotic change to occur in the aorta before it would lead to dilatation and the risk of rupture.
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> . . . .
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> In their younger years fire fighters are selected because of their good health so that they are able to tolerate these stresses but as they get older and their physical fitness may not be as good, the stresses may become greater. The main question is really whether chronic stresses and chronic toxic exposure predisposes them to aortic aneurysm or other cardiac conditions. I am unaware of any chronic effect of carbon monoxide. Its effects should

> be acute. Some of the other chemicals could conceivably have some acute or possibly chronic affect.
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> . . . .
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> Early studies [of the effects of cigarette smoking] suggested that carbon monoxide might be a causative agent however these have not been confirmed. Becker identified agents derived from cigarette smoke that may be injurious to the arterial wall. Similar comments might be made with respect to periodic smoke inhalation by fire fighters." [208]

Dr. C. Morgan raised a number of concerns about the validity of the aortic aneurysm finding in the IDSP study, including the potential inaccuracy of death certificate data, the small number of aortic aneurysm deaths and the problem of multiple tests of significance. He stated that:

> " ... although potentially real, the finding of excess death secondary to aortic aneurysm must be considered as speculative at best. The overall risk of atherosclerotic cardiovascular disease does not appear to be substantially increased amongst firefighters. As aortic aneurysm and ischemic heart disease, for example, share a common etiology and risk factors it is difficult to identify any biological process which might selectively increase the risk of aortic aneurysm, independent of other manifestations of atherosclerosis.
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> . . . .
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> Exposure to carbon monoxide is a potential cause of accelerated atherosclerosis for which there is some experimental support. The finding of no excess in total or cardiovascular death amongst firefighters in many of the studies summarized in the literature review suggests that this or other exposures may not have a major cumulative adverse cardiovascular effect." [126]

Dr. Melius, however, noted that the excess of deaths from aortic aneurysms was statistically significant in the Toronto firefighters and said:

> "One possible explanation would be a factor related to fire fighting which increased the process of atherosclerosis. The increased risk of other cardiovascular disease in firefighters ... suggests that fire fighting may increase the risk of atherosclerosis (in turn leading to increased mortality from cardiovascular

> disease). There is some scientific evidence that exposure to carbon monoxide increases the risk of developing atherosclerosis. This includes experimental evidence (included in your literature review) as well as the increased risk of cardiovascular disease found in cigarette smokers. Studies of workers with high exposures to carbon monoxide have found an increased risk of cardiovascular disease (Stern et al. study of bridge and tunnel workers). This atherosclerotic effect is different from the more direct metabolic effect from carbon monoxide which may contribute to the occurrence of myocardial infarctions or angina in people exposed to relatively low levels of carbon monoxide. Epidemiological studies of cigarette smokers have shown that their higher risk of dying of cardiovascular disease falls off after they cease smoking, but some increased risk does persist ... In summary, while I am not aware of any other evidence of an increased risk of aortic aneurysm in fire fighters, the increased mortality found in this study is plausible based on evidence linking carbon monoxide to an increased risk of atherosclerosis and the increased exposure of fire fighters to carbon monoxide.
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> . . . .
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> I am not aware of any consistent evidence that other components of fire smoke may increase the risk of arteriosclerosis. However, fire smoke has not been well characterized and could contain such substances.
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> . . . .
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> Dietary and genetic factors are unlikely to account for the excess risk [in arteriosclerosis] seen in the epidemiological study given the comparison with the general population, the number of cases, etc." [119]

The Panel also asked an Epidemiological Consultant with expertise in the causes and development of aortic aneurysms to comment on this statistically significant finding. Dr. D. Reed advised the Panel that:

> "It is generally accepted that damage to or defects in aortic wall matrix proteins are required to develop an aneurysm ... and anything that would reduce the flow of

> nutrients could lead to deterioration of the protein architecture of the aorta wall.
>
> In considering occupational factors which could affect this process, smoke is the first suspect. As noted in the enclosed paper, cigarette smoking was the strongest predictor of aortic aneurysms ... Possible causal mechanisms include direct endothelial damage with thickening of the endothelial wall, platelet aggregation or vasoconstriction which would obstruct blood flow in the vasa vasorum, and the atherogenic role of smoking on the aorta. High levels of carbon monoxide, and decreased oxygen levels associated with airway obstruction could add to this causal process. Thus I think that there are biologically plausible causal factors for an increased risk of aortic aneurysm among fire fighters.
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> . . . .
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> In regard to your question concerning some type of exposure which would increase the risk of aortic aneurysm independently from the risk of death due to other cardiovascular [causes], there is also some information to consider. It has been well documented that atherosclerosis in the aorta precedes that in the coronary arteries by about 20 years. Furthermore, the role of cigarette smoking has a much stronger association with atherosclerosis in the aorta than in the coronary arteries ... Aortic atherosclerosis has also been found to be disproportionately severe in countries with low incidence rates of coronary heart disease ... Exposure to indoor cooking and heating with open fires has been suggested as one of the reasons for this finding. Thus it is quite possible that there are factors affecting the aorta which have a delayed or lesser effect on other vessels." [157]

Another possible risk factor identified in the study by Dr. Reed and co-workers (1992) is height. Aortic aneurysms were associated to a statistically significant degree with height in that study and in another small group of patients with atherosclerotic occlusive disease reported by Tilson and Dang (1981). Reed and colleagues wrote:

> "One possible explanation of this association is that large persons have large arteries, which in turn have greater circumferential wall stress (force per unit area) as described by the physical law of Laplace (tension is equal to pressure multiplied by radius)."[158]

In that study, the risk factors that predicted aortic aneurysms were high blood pressure, high serum cholesterol, cigarette smoking and height. Height was the only risk factor that was not also independently associated with aortic atherosclerosis. In other words, height was a risk factor for aortic aneurysm, but not for atherosclerosis [158].

A study of Toronto firefighters by Bates notes an unpublished survey showing that in 1980 firefighters were smoking at about the same rate as the rest of Canadians, that is, about one-third of Toronto firefighters smoked [8]. This suggests that smoking is not a significant confounder.

- **Other findings of elevated mortality due to aortic aneurysm**

Stern and colleagues (1980) examined the mortality experience of 1558 white male motor vehicle examiners exposed to carbon monoxide and found

> " ... a slight excess of deaths in the 'Diseases of the Cardiovascular System' category [124 observed (obs.) vs. 118.4 expected (exp.)]. Within this category, there was a deficit of deaths due to arteriosclerotic heart disease (89 obs. vs. 96.4 exp.), but a statistically significant excess of deaths due to diseases of the arteries and veins (13 obs. vs. 5.9 exp.; $p<.05$). More specifically, when aneurysms of the heart and syphilitic[15] aneurysms of the aorta were excluded, a statistically significant excess was observed in mortality due to aneurysms of all other sites (7 obs. vs. 2.1 exp.; $p<.05$)."

Dr. Stern advised the Panel that four of the seven observed aneurysms of "all other sites" were non-syphilitic aortic aneurysms (ICD-7 code #451). Unfortunately, expected figures for non-syphilitic aortic aneurysms specifically are not available but it is clear that mortality due to non-syphilitic aneurysm was at least twice the expected [186, 187].

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15 Dr. John Wilson advised the Panel that in days gone by, syphilitic infection caused aneurysm of the ascending aorta but this is rarely seen since the introduction of an effective treatment for syphilis[208].

Discussion

In all, findings for aortic aneurysm from five firefighter studies were available to the Panel. A statistically significant increase occurred for the whole cohort in the IDSP study. In two studies, subsets of long-serving firefighters experienced elevations in mortality which were also statistically significant. Of these five studies, only one did not identify any statistically significant increases.

Four of the studies' findings did not correct for the potential underestimation in risk attributable to the healthy worker effect.

Summary of the evidence regarding aortic aneurysm

- The statistically significant increase in aortic aneurysms among firefighters compared to the age-matched general population of Ontario found in the IDSP study is not attributable to multiple significance testing and is persuasive of a probable connection between aortic aneurysm and the occupation of firefighter.

- The importance of this increase is confirmed by similar findings for groups of long-serving firefighters in other mortality studies.

- There is a lack of coherence between the rates of mortality due to aortic aneurysm and other cardiovascular outcomes. This may be attributable to the occurrence of aortic aneurysms well prior to other manifestations of atherosclerosis, as advised by Dr. Reed.

- There is evidence that carbon monoxide, which is a by-product of all fire, accelerates arteriosclerotic changes, as demonstrated in some research on animals. Cigarette smoking was the strongest predictor of aortic aneurysm in Dr. Reed's research and there is some evidence to suggest that this is due to the carbon monoxide in cigarette smoke. Motor vehicle examiners exposed to carbon monoxide experienced a significant increase in mortality due to aortic aneurysm.

- Aortic aneurysms are also linked to smoking, but since there is no evidence that Toronto firefighters smoked any more than the general Ontario male population to which they were compared, smoking is unlikely to explain the more than doubling of the risk.

- **The Panel's conclusions and finding regarding aortic aneurysms**

The Panel finds that the evidence in support of an association between firefighting and aortic aneurysm is persuasive because of the statistically significant excess found among Toronto firefighters, a finding which cannot be dismissed as the result of multiple tests of significance. There is some consistency in support of an association in the statistically significant increases found among long-serving firefighters from two other cohorts. By analogy, a statistically significant excess was found among motor vehicle examiners who, like firefighters, are exposed to carbon monoxide. Finally, the Panel is advised that the exacerbation of atherosclerosis by carbon monoxide which results in aortic aneurysm prior to other cardiovascular manifestations is biologically plausible. Accordingly, the Panel makes the following finding.

- **A probable connection exists between firefighting and atherosclerosis which results in aortic aneurysm.**

Because of the strength of the evidence of a probable connection between firefighting and aortic aneurysm, the Panel concludes that the disease and the process be included in a Schedule appended to the *Workers' Compensation Act*. The fact that aortic aneurysms also occur in the general population due to non-occupational causes has persuaded the Panel that the entry should be included in Schedule 3, and not in Schedule 4.

This recommendation would give a firefighter a presumption that the aortic aneurysm was due to employment, unless the contrary is proved. This would mean that such a firefighter would receive workers' compensation benefits unless it could be proven that the aneurysm was not related to firefighting. Evidence which might prove the contrary could include information about latency, alternative causes or duration of employment as a firefighter. To allow the WCB adjudicators to fairly and equitably assess the evidence that would be used to rebut the presumption, it is critical to develop guidelines or a rebuttal matrix.

The Panel's recommendations regarding aortic aneurysm

- **"Atherosclerosis which results in aortic aneurysm" and the associated process, trade or occupation of "firefighter" should be added to Schedule 3 of the *Act*.**

- **A rebuttal matrix approved by the Panel should be used to assess the evidence used to rebut the presumption.**

TABLE 10: FIREFIGHTER MORTALITY/MORBIDITY STUDIES: CARDIOVASCULAR DISEASE FINDINGS

AUTHORS; YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	CARDIO-VASCULAR - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
ORRIS, et al.; (1992); Proportional Mortality Ratio ("PMR") study	3084 white male FF deaths	1940-88 (49 yrs.)	U.S.A. white males	Chicago, Illinois, U.S.A.	not applicable	not applicable	Ischemic Heart Disease: PMR 111; (106-118) Other Circulatory Diseases: PMR 82; (73-93); No dose-response analyses.	Analysis done for IDSP: Adjusted PMR for aortic aneurysms during 1953-1957: 3.115 ($p<0.05$); (95% C.I. 1.142-6.697)
MUSK et al.; (1978); Standardized Mortality Ratio ("SMR") study	5655 males	1915-75 (61 yrs.)	male population of Mass. and U.S.A.	Boston, Mass., U.S.A.	yes	2470 deaths; 91% of expected for Mass. males; 94% of expected for U.S.A. white males	Circulatory, overall: SMR 86; (81-91); n=1058; no dose-response analyses. Findings do not show mode of death in subjects dying from cardio-vascular disease.	
L'ABBÉ & TOMLINSON (1992); (IDSP study); Standardized Mortality Ratio ("SMR") study	5,414 males; 5,373 for duration of employment analysis	1950-89 (40 yrs.)	Ontario males	Metropolitan Toronto	strong for FFs under 40 and for FFs with less than 10 years since first exposure	SMR 94; (88-101); n=777	Aortic Aneurysm: SMR 226; (136-353); n=19; possible dose-response. Arteriosclero-sis: SMR 141; (91-210); n=24; no dose-response; Ischemic: SMR 104; (92-117); n=289; no dose-response; Circulatory overall: SMR 99; (89-109); n=384; no dose-response.	

AUTHORS; YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	CARDIO-VASCULAR - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
DEMERS et al.; (1992); Standardized Mortality Ratio ("SMR") study	4546 males	1945-89 (45 yrs.)	1) U.S. white males; 2) 3676 police from Seattle & Tacoma, Wash., U.S.A. (white males)	Seattle & Tacoma, Wash, U.S.A.	yes, except for diseases of the arteries, veins & pulmonary circulation in FFs with 30 or more years of exposure	SMR 0.81; (0.77-0.86); n=1169	Heart: SMR 79; (72-87); n=461; IDR compared to police 86; (74-100); Ischemic: SMR 0.82; (0.74-0.90); n=394; IDR compared to police 0.88; (0.74-1.04); Other Circulatory: SMR 96; (80-114); n=131; IDR compared to police 72; (54-96); no dose-response analyses for these causes; Diseases of veins, etc. increases with latency, possible survivor effect, but no dose-response	compares FFs with police, who are also screened for physical health; Analysis done for IDSP: Aortic aneurysm SMR among NW US FFs employed 30+ yrs.: 2.47 (1.13-4.69); n=9; no statistically significant increase among Toronto FFs compared to NW US police
GUIDOTTI; (1993); Standardized Mortality Ratio ("SMR") study	3328	1927-87 (61 yrs.)	Alberta males	Edmonton & Calgary, Alberta	no, probably because: 1-Alberta males are unusually healthy; and, 2-most subjects were traced	SMR 96.2; (86.6-106.5); n=370	Circulatory overall: SMR 103.1; (87.6-120.6); n=157; Ischemia: SMR 105.9; (86.9-127.7); n=109; Heart: SMR 110.4; (92.2-131.1); n=130; Arteriosclero-sis: SMR 149.7; (77.3-261.5); n=12; no dose-response for "cardiovascular disease"	Analysis done for IDSP: 2 deaths from aortic aneurysm; both occurred between 1985-87 but had no other common features.
BEAUMONT et al.; (1991); Mortality Rate Ratio ("RR") study	3066 white males	1940-82 (43 yrs.)	U.S.A. white males	San Francisco, Calif., U.S.A.	yes	RR 0.90; ($p<0.05$); (0.85-0.95); n=1186	Heart: RR 0.89; ($p<0.05$); (0.81-0.97); n=508;dose-response not apparent; Ischemic: RR 0.95; (0.87-1.04); n=457; no dose-response analysis for this cause	

AUTHORS; YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	CARDIO-VASCULAR - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
HEYER et al.; (1990); Standardized Mortality Ratio ("SMR") study	2289 males	1945-83 (39 yrs.)	U.S.A. white males	Seattle, Wash., U.S.A.	yes; survivor effect also noted.	SMR 76; (69-85); n=383	Circulatory: SMR 78; (68-92); n=172; dose-response shown. Relative risk of death was 1.84 in FFs with 30+ years of exposure compared to those with less than 15 years exposure; Arteriosclero-tic: SMR 75; (63-89); n=133; no dose-response analysis for this cause	Authors suggest this may be due to repeated exposure to high levels of carbon monoxide.
VENA & FIEDLER; (1987); Standardized Mortality Ratio ("SMR") study	1867 white males	1950-79 (30 yrs.)	U.S.A. white males	Buffalo, N.Y., U.S.A.	yes, especially for circulatory system diseases.	SMR 0.95; (0.87-1.04); n=470	Circulatory, overall: SMR .92; (.81-1.04); n=250; Arteriosclerotic: SMR .92 (.79-1.07); n=173; clear dose-response shown for both	Analysis done for IDSP: Aortic aneurysm: SMR 163; (65-337); n=7; possible small dose-response
TORNLING et al.; (1994); Standardized Mortality Ratio ("SMR") study	1116	1951-86 (36 yrs.)	Stockholm males	Stockholm, Sweden	yes	SMR 82; (73-91); n=316	Circulatory, overall: SMR 84; (71-98); n=161; Ischemic: SMR 98; (81-117) n-118; dose-response not apparent; increases with latency	
DECOUFLE et al., (1977); Proportionate Mortality Ratio ("PMR") study	1113 stationary engineers and firefighters	1967 (1 year)	U.S. PMRs	4 areas of U.S.	not applicable	not applicable	Heart disease, overall: PMR 1.05; (0.96-1.14); n=527; Coronary heart disease: PMR 1.06; (0.96-1.16); n=450; no dose-response analyses	combines findings for engineers and firefighters which may dilute or overesti-mate results
MASTRO-MATTEO; (1959); Standardized Mortality Ratio ("SMR") study	1039	1921-53 (33 yrs.)	Toronto & Ontario males	Toronto, Ontario	not mentioned	significant excess of deaths for all causes compared to Toronto males	Cardio-vascular-Renal Deaths: Significantly higher than both Toronto & Ontario males; no dose-response analyses	

AUTHORS; YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	CARDIO-VASCULAR - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
ELIOPULOS et al.; (1984); Standardized Mortality Ratio ("SMR") and Proportionate Mortality Ratio ("PMR") study	990	1939-78 (about 40 yrs.)	Western Austr. males	Western Australia	yes	SMR 0.80; (0.67-0.96); n=116	Circulatory, overall: SMR .78; (.60-1.01); n=55; Ischemic: SMR .84; (.60-1.14); n=39; no dose-response analyses.	
HANSEN; (1990); Standardized Mortality Ratio ("SMR") study	886	1970-80 (10 yrs.)	Danish civil servants	Denmark	unlikely	SMR 99; (75-129); n=57	Ischemic Heart Disease: SMR 115; (74-171); n=24; no dose-response analyses; Mortality from ischemic heart disease was increased only in the first 5-year period.	
BATES; (1987); *Coronary artery disease* Standardized Mortality Ratio ("SMR") study	646	1949-1959 (11 years)	City of Toronto males	Toronto, Ontario	yes	not given	Coronary artery disease: SMR 1.73; (1.12-2.66); n=21; no dose-response	Coronary artery disease among ages 45-49 between 1975-79: SMR 2.47;($p<0.10$)
SARDINAS et al; (1986); Standardized Mortality Ratio ("SMR") and Odds Ratio ("OR") study	306 FF deaths	1960-78 (19 yrs.)	1) deaths among state workers; 2) 401 police deaths	state of Connecticut, U.S.A.	yes	not given	Ischemic Heart Disease: compared to state workers: SMR 1.52; (1.23-1.81); n=115; OR 1.07; (0.91-1.23); compared to police: OR 0.62; (0.56-0.68); no dose-response analyses	

AUTHORS; YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	CARDIO-VASCULAR - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
FEUER & ROSENMAN; (1986); Proportionate Mortality Ratio ("PMR") study	263 FF deaths	1974-80 (7 yrs.)	1) U.S. PMRs; 2) N.J. PMRs; 3) N.J. police PMRs	state of New Jersey, U.S.A.	not applicable	not applicable	Arteriosclero-tic heart disease: compared to U.S. males: PMR: 1.22; (1.01-1.46); ($p<0.05$) compared to New Jersey males: PMR: 1.11; (0.92-1.33); compared to police: PMR: 1.06; (0.88-1.27); n=115; no dose-response Circulatory: compared to U.S. males: PMR: 1.09; (0.91-1.29); compared to New Jersey males: PMR: 1.02; (0.85-1.21); compared to police: PMR: 1.01; (0.84-1.20); n=131; no dose-response analysis for this cause	compares FFs with general U.S. and state popula-tions and with police, who are also screened for physical health
GRIMES et al.; (1991); Proportionate Mortality Ratio ("PMR") study	205 males	1969-88 (20 yrs.)	20 year and older Hawaiian males	Honolulu, Hawaii, U.S.A.	mentioned by authors	not applicable	All circulatory: RR 1.16; (1.10-1.32); Arteriosclero-tic heart disease: RR 1.09; (0.89-1.35); no dose-response analyses.	
MILHAM; (1983); Proportionate Mortality Ratio ("PMR") study	not stated	1950-79 (30 yrs.)	Washington state males	state of Washington, U.S.A.	not applicable	not applicable	Circulatory, overall: PMR 101; (94-108); n=777; Arteriosclerotic heart disease including coronary disease: PMR 105; (97-114); n=622; Diseases of veins, etc.: PMR 131; (65-234); n=11; no dose-response analyses	

f) Cancer among firefighters

(i) Parameters of this Report

One of the issues originally raised by the firefighters' representatives was whether a legal presumption should apply to claims for lung cancer from firefighters. Whether a probable connection exists between lung cancer and firefighting will be the first issue addressed, however, the Panel did not limit its investigations to lung cancer.

The Panel considered the cancer findings of all the available firefighter studies including the IDSP study. A set of criteria was needed to organize this large amount of data and ensure that associations that might exist were not overlooked. These wide parameters do not necessarily indicate findings of association but were used by the Panel to structure its detailed deliberations.

- A statistically significant SMR (or PMR, OR or RR) reported in any large firefighter mortality or morbidity study; that is, an SMR of 100 or higher where the lower end of the 95% confidence interval is 101 or more; or
- An SMR (or PMR, OR or RR) of 170 or higher regardless of the confidence interval; or
- A lower end of the 95% confidence interval between 90 and 100; or
- A dose-response trend indicated; or
- A disease about which the firefighters' representatives expressed particular concern, such as lung cancer; or
- Diseases to which the state of Massachusetts has applied a statutory presumption if at least one firefighter study found a statistically significant increase in risk (discussed under "Policy and claims experience considerations regarding cancer", above).

In accordance with these parameters, the Panel examined in detail the findings from the IDSP study and other studies for the following cancer types.

- lung cancer
- brain cancer
- lymphatic and haematopoietic cancers
- colon cancer
- bladder cancer
- kidney cancer
- rectal cancer

As discussed under "Policy and claims experience considerations regarding cancer" above, a rebuttable presumption applies to claims for skin cancer from workers who have been exposed to "tar, pitch, bitumen, mineral oil or paraffin or any compound, product or residue of any of these substances" pursuant to entry 4 in Schedule 3 of the Regulations to the *Act*. There is little doubt that fighting fires involves exposure to these substances, since they are widely used in construction materials, etc., hence a rebuttable presumption applies to such claims from firefighters. Accordingly, skin cancer among firefighters will not be discussed further in this Report.

Finally, the Panel considered the distinctions between mortality and morbidity data when used to assess the risk of occupational disease. Dr. Norman Boyd, in his letter of August 28, 1992, advised the Panel that

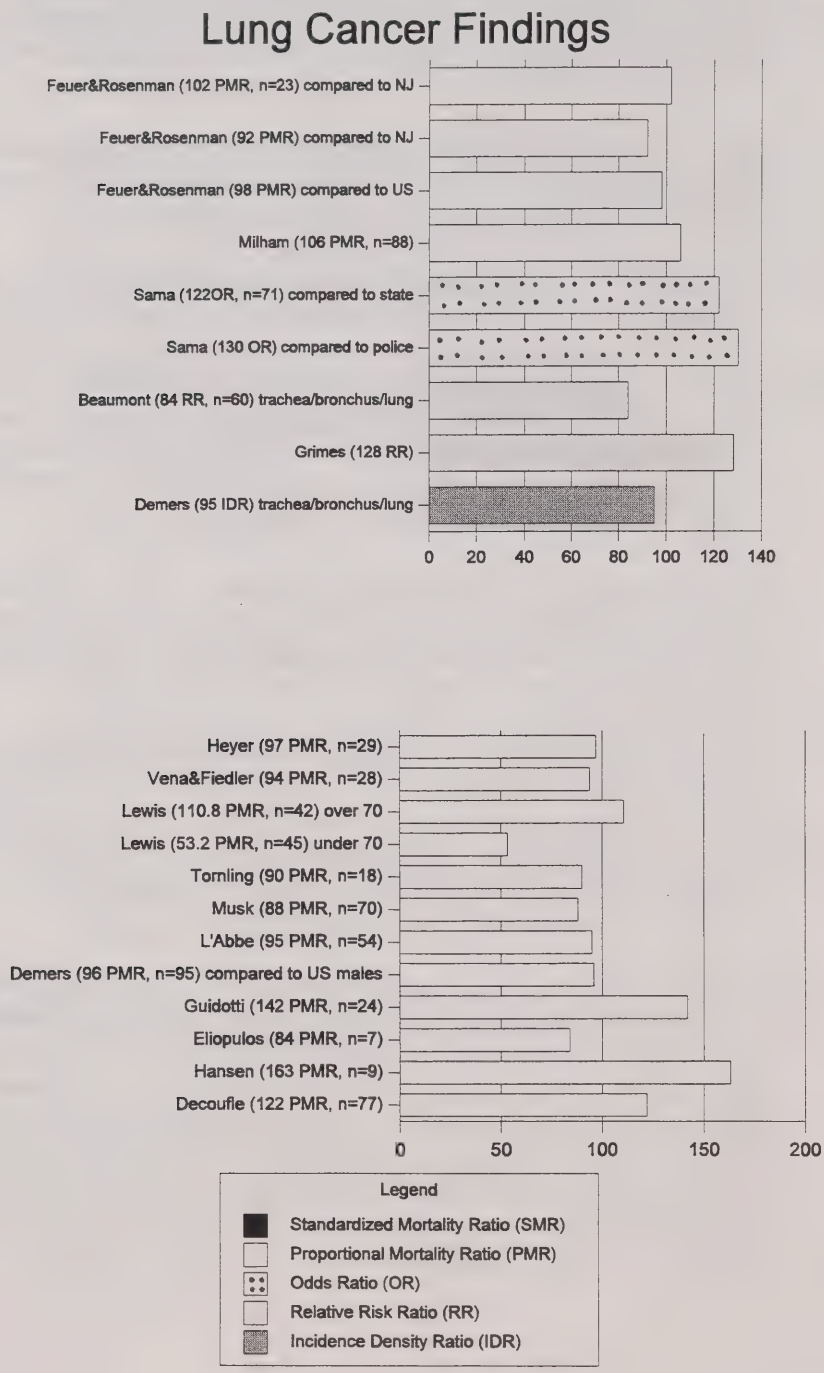
> "Cancers for which morbidity information would be preferable to mortality include all the lymphatic and haematopoietic sites, and testis cancer. In these diseases a substantial proportion of those affected will not die of the disease because curative treatment is available. Prolonged survival is possible in several other sites, such as prostate, rectum and melanoma and incidence data would be preferred. Incidence data is also based upon pathology reports and is likely to be more reliable for all sites than mortality data based upon death certificates." [17]

With the assistance of the Ontario Cancer Treatment and Research Foundation [140], the Panel attempted to conduct an incidence analysis of the data from its Mortality Study of Firefighters in Metropolitan Toronto. This analysis proved to be problematic with the data that was available to the IDSP.

The mortality data provided by Statistics Canada from the National Mortality Data Base was not provided in a format that identified individual firefighters. To be able to determine the person-years at risk for an incidence study it would have been necessary to regenerate the data in a format that contained personal identifiers. The Panel considered this option and declined to undertake these additional steps for several reasons.

Firstly, the IDSP had a consultant perform an analysis of the data using two different methods to estimate the person-years at risk. The results when applying either method for the same types of cancer did not contradict the findings of the mortality study. Furthermore, since the commencement of this project many mortality studies and at least one large incidence study of firefighters have been completed. This has resulted in a significant body of evidence about the health effects of firefighting. It was the opinion of the Panel that it was unnecessary to conduct yet a further study to have sufficient evidence to complete its deliberations.

Fig. 7


Lung Cancer Findings
Feuer&Rosenman (102 PMR, n=23) compared to NJ
Feuer&Rosenman (92 PMR) compared to NJ
Feuer&Rosenman (98 PMR) compared to US
Milham (106 PMR, n=88)
Sama (122OR, n=71) compared to state
Sama (130 OR) compared to police
Beaumont (84 RR, n=60) trachea/bronchus/lung
Grimes (128 RR)
Demers (95 IDR) trachea/bronchus/lung
0 20 40 60 80 100 120 140
Heyer (97 PMR, n=29)
Vena&Fiedler (94 PMR, n=28)
Lewis (110.8 PMR, n=42) over 70
Lewis (53.2 PMR, n=45) under 70
Tomling (90 PMR, n=18)
Musk (88 PMR, n=70)
L'Abbe (95 PMR, n=54)
Demers (96 PMR, n=95) compared to US males
Guidotti (142 PMR, n=24)
Eliopulos (84 PMR, n=7)
Hansen (163 PMR, n=9)
Decoufle (122 PMR, n=77)
0 50 100 150 200
Legend
Standardized Mortality Ratio (SMR)
Proportional Mortality Ratio (PMR)
Odds Ratio (OR)
Relative Risk Ratio (RR)
Incidence Density Ratio (IDR)

(ii) Lung cancer

It is estimated that lung cancer accounted for 20.5% of new cancer cases and 33.5% of cancer deaths among adult males in Canada in 1993 [130].

Consultants' comments

None of the three medical experts consulted by the Panel thought there was a clear link between firefighting and lung cancer.

Dr. Quirt noted that lung cancer is extremely common and said, "The studies do not point to an increased risk of lung cancer in firefighters when compared to the normal population." [155]

Dr. McDiarmid also acknowledged that neither the IDSP study's findings nor those of other studies indicate an association between firefighting and lung cancer. She added:

> "However, non-malignant respiratory disease has consistently been found among this working cohort [47, 181]. Issues which have been raised to explain this include the following: a) Firefighters may exit the fire service prior to cancer development much as smokers who may quit prior to disease development. In the US, firefighters may be eligible for retirement after twenty years. It is also possible that twenty years of exposure is not a long enough exposure for the majority of firefighters to develop lung cancer and they therefore retire prior to their excess risk being manifested. b) A firefighter who develops non-malignant respiratory disease may self-select out of the fire service. A firefighter who is suffering from the irritation of chronic obstructive pulmonary disease may find the firefighting environment too irritating to tolerate and may leave the fire service." [111]

The Panel agrees that some firefighters may be able to leave the profession early enough to escape developing occupational lung cancer. Other firefighters may develop lung cancer only after they have retired from the profession. Since most of the studies included retired firefighters, however, it is unlikely that an excess of lung cancer occurred among retired firefighters and was obscured.

The issue of occupational non-malignant respiratory disease among firefighters will be the subject of a subsequent Panel Report.

FIG. 8: LUNG CANCER MORTALITY AMONG DANISH FIREFIGHTERS, NOVEMBER 9, 1975, TO NOVEMBER 9, 1980, BY AGE:

AGE AT NOVEMBER 9, 1975:

Age	SMR	(95%CI)
30-49	0	(0-149)
50-59	135	(16-488)
60-74	317	(117-691)
Total	220	(95-434)

(Hansen, 1990)

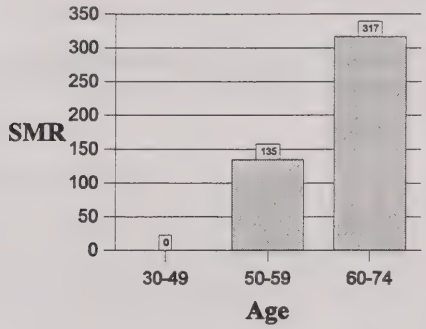
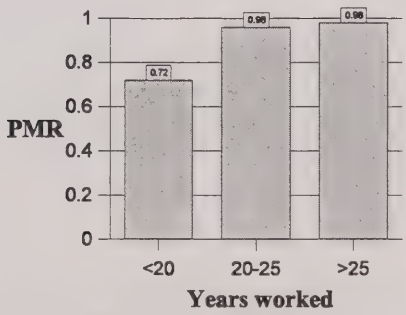


FIG. 9: RESPIRATORY CANCER MORTALITY AMONG NEW JERSEY FIREFIGHERS, 1974-1980, BY DURATION OF EMPLOYMENT:

Years worked	PMR	(95%CI)	n
<20	0.72	(20-184)	4
20-25	0.96	(39-198)	7
>25	0.98	(51-171)	12

(Feuer and Rosenman, 1986)
n=number of deaths



Mortality and morbidity studies

Lung cancer was elevated, but not to a statistically significant degree, in six of seventeen studies which gave findings for firefighters' mortality or morbidity. Two of those studies, however, by Hansen (1990) and Heyer et al. (1990), identified increases which were statistically significant in subgroups of older firefighters. One author reported a statistically significant decrease in firefighters under age 70 [98]. Three found a possible dose-response trend, four did not, and ten did not conduct dose-response analyses for this disease.

Findings from the epidemiological studies are illustrated in Fig. 7. More detail is provided in Table 11 which appears at the end of this section.

Statistically significant increases

Heyer and colleagues' (1990) study of mortality among 2289 Seattle firefighters found a statistically significant excess of lung cancer deaths among firefighters over 65 years of age (SMR 177; 95% CI 105-279), but not in younger men compared to the general population.

A statistically significant excess in lung cancer mortality also occurred in the 60-74 age group of Danish firefighters (Fig. 8) studied by Hansen (1990).

Statistically significant decrease

Lewis and co-workers (1982) found a statistically significant decrease in lung cancer mortality among Los Angeles firefighters under 70 years of age (SMR 53.2; (95% CI 39-71); n=45). For those over 70, the SMR was 110.8 (95% CI 80-150; n=42).

Other findings

A 1986 New Jersey report by Feuer and Rosenman (1986) on the causes of 263 firefighter deaths found fewer than expected from respiratory cancer compared with the US and state populations. The rate was slightly higher than expected, however, when firefighters were compared with state police, who also experienced fewer lung cancer deaths than the US and state populations. A small dose-response trend appeared (Fig. 9), although the PMR for those with more than 25 years of employment was only 0.98.

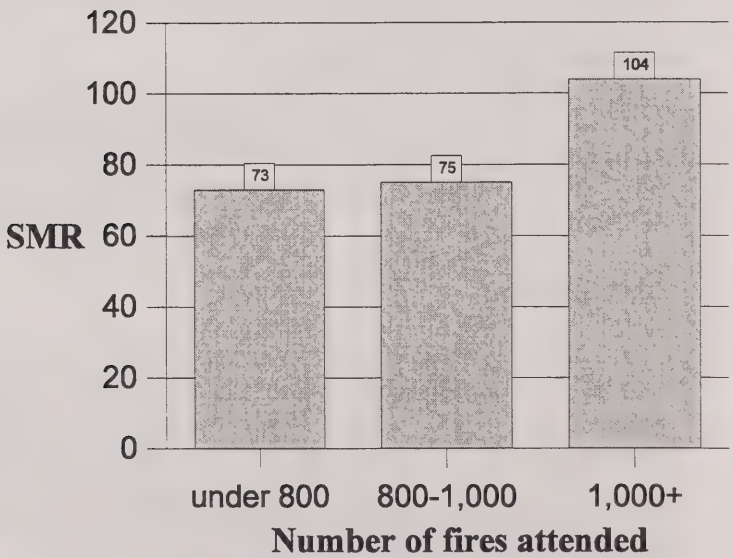
A case-control study that involved 321 cases of cancer among Massachusetts firefighters by Sama and colleagues (1990) compared firefighters with police and the general population from that state. The authors found elevations in all lung cancer rates which did not reach statistical significance for firefighters. As in the New Jersey study, the elevations were higher compared to police than compared to the state population.

FIG. 10: LUNG CANCER MORTALITY AMONG SWEDISH FIREFIGHTERS, 1951-1986, BY NUMBER OF FIRES ATTENDED:

Fires attended	SMR	(95%CI)	n
under 800	73	(15-214)	3
800-1,000	75	(20-192)	4
1,000+	104	(52-185)	11

n=number of deaths

(Tornling et al., 1990)



That was the only study in which smoking information was considered. More Massachusetts firefighters than police or state males were current smokers and fewer were former or never smokers. The authors did not consider the elevation in firefighters' lung cancer rate noteworthy given an SMR of 122 compared to state males and 130 compared to police [169]. Brackbill and colleagues (1988) analyzed data from the 1978-1980 US National Health Interview Survey and reported that about the same proportion of firefighters (44.5%) and police (45.2%) were current smokers, but 42.1% of firefighters had never been smokers, compared to only 29.9% of police.

One other study compared firefighters with police. Demers and colleagues [1992] found slight reductions, which were not statistically significant, in lung cancer deaths among 4546 Northwestern US firefighters compared with both police and the US population [Demers, 1992].

Dose-response analyses

Of seven studies which provided a dose-response analysis for lung cancer, two showed an increasing trend. Tornling and co-workers (1994) noted increasing SMRs with the number of fires attended (Fig. 9), although the SMR for those who attended more than 1000 fires was not appreciably raised (SMR 104; 95% CI 52-185; n=1). A dose-response trend was also shown in the New Jersey cohort studied by Feuer and Rosenman (1986) (Fig. 8).

The IDSP study noted a slightly decreased rate of lung cancer deaths among 5414 Toronto firefighters compared to Ontario males (SMR 95; 95% CI 71-123; 54 deaths) and no dose-response trend [86].

Potential causative agents

In addition to the recognized link between smoking and lung cancer, there is evidence to support an association between lung cancer and the following airborne contaminants of fire smoke: vinyl chloride, asbestos, soots, PAHs, diesel exhaust, acrylonitrile and formaldehyde [79, 80, 81, 176], to which firefighters are very likely exposed. There is evidence that firefighters are also exposed to lung irritants.

Vinyl chloride

As a primary ingredient in the manufacture of many plastic articles, vinyl chloride vapour may be released during the thermal decomposition of these articles when they are sufficiently heated. Small amounts of vinyl chloride have been measured in the smoke of fires involving plastic materials [106].

Asbestos

None of the studies reviewed included asbestos in their analyses of airborne contaminants at the fire site. Since asbestos was widely used in the manufacture of various building insulation materials, it is anticipated that firefighters may be significantly exposed to airborne asbestos fibres at fires where such materials are present. Markowitz et al. (1980) reported evidence that firefighters are at risk for scarring of the lungs and pleura due to occupational asbestos exposure.

Soots and PAHs

IARC notes statistically significant excesses in mortality from scrotal and other cancers among chimney sweeps exposed to soots (Group 1) [79]. It is likely that firefighters are also significantly exposed during fire fighting since all fires generate soots.

Soots contain PAHs, many of which are known carcinogens to humans. These particulates have been measured in detectable amounts in the smoke of building fires [82].

Diesel exhaust

Firefighters are exposed to PAHs in diesel exhaust when engines are started in fire stations [49].

Acrylonitrile and formaldehyde

Both acrylonitrile and formaldehyde may be present in the air at fire sites as combustion products of building and insulation materials. None of the studies reviewed included acrylonitrile in their chemical analyses of fire smoke; however, it is anticipated that firefighters could be frequently exposed to acrylonitrile vapours generated from thermal decomposition of various rubber products. As for formaldehyde, significant concentrations have been measured in studies of different types of fires [19, 82].

Other information

Neither the IDSP literature review nor that conducted for the British Columbia WCB concluded that there is an increased risk of lung cancer among firefighters [222, 74].

Summary of the evidence

- Although statistically significant increases in lung cancer mortality occurred in two subgroups, none of the studies identified an overall increase which reached statistical significance.

- One author identified a statistically significant decrease in lung cancer deaths.

- Of seven studies which provided a dose-response analysis for lung cancer, three were suggestive of such a trend and four were not. Again, the Panel concludes that the use of duration of employment as a surrogate for exposure does not provide meaningful information about "dose", hence the lack of a dose-response trend is also not meaningful.

- There is evidence to support a link between lung cancer and many of the substances to which firefighters may be exposed (asbestos, vinyl chloride, diesel exhaust, soots, acrylonitrile, PAHs and formaldehyde). There is also evidence that other firefighter exposures are lung irritants.

The Panel's conclusions and finding

The evidence about lung cancer was one of the surprising findings. There is substantial evidence that firefighters are exposed to agents that are known to cause lung cancer. It is also acknowledged that SCBA has not been completely effective in preventing the inhalation of carcinogens. This is particularly true of asbestos which would be present during the overhaul phase of many fires. For these reasons, the Panel anticipated that there would be evidence of excess lung cancer among firefighters; however, that proved not to be the case.

As mentioned previously, the Panel uses the guidelines developed by Sir Austin Bradford Hill to assess the evidence of probable connection. The absence of some evidence should not be taken to disprove an association. A lack of evidence is simply uninformative; however, when a causal relationship is explored repeatedly and not found, as in the case of lung cancer and firefighting, a probable connection can be said not to exist.

The Panel therefore makes the following finding.

- **There is not a probable connection between lung cancer and the occupation of firefighting.**

The Panel's recommendation

There may be circumstances in which a firefighter contracts lung cancer due to work. Accordingly, the Panel makes the following recommendation.

- **The Panel recommends that the WCB continue to adjudicate claims for lung cancer from firefighters having regard for the individual merits of each claim.**

TABLE 11: FIREFIGHTER MORTALITY/MORBIDITY STUDIES: LUNG CANCER FINDING

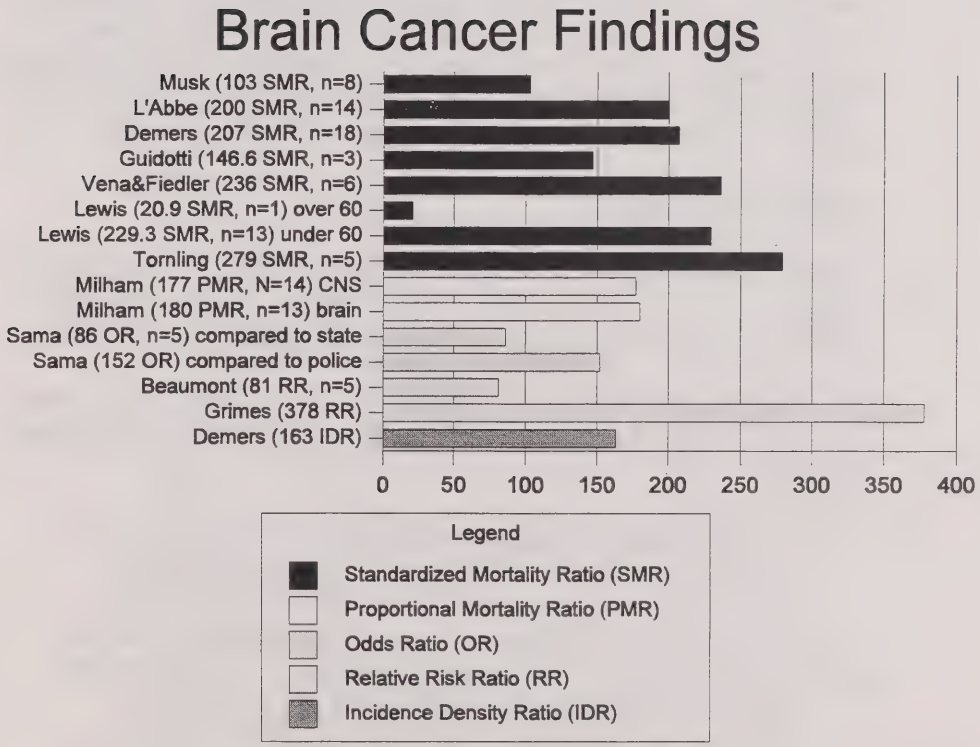
AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	LUNG CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
MUSK et al.; (1978); standardized mortality ratio ("SMR") study	5655 males	1915-75 (61 yrs.)	male population of Mass. and U.S.A.	Boston, Mass., U.S.A.	yes	2470 deaths; 91% of expected for Mass. males; 94% of expected for U.S.A. white males	Lung cancer: SMR 88; (69-111); n=70; no dose-response analysis	
L'ABBÉ & TOMLINSON; (1992); (IDSP study); standardized mortality ratio ("SMR") study	5,414 males; 5,373 for duration of employment analysis	1950-89 (40 yrs.)	Ontario males	Metropolitan Toronto	strong for FFs under 40 and for FFs with less than 10 years since first exposure	SMR 94; (88-101); n=777	Trachea, bronchus and lung cancer: SMR 95; (71-123); n=54; dose-response not apparent	
DEMERS et al.; (1992); standardized mortality ratio ("SMR") study	4546 males	1945-89 (45 yrs.)	1) U.S. white males; 2) 3676 police from Seattle & Tacoma, Wash., U.S.A. (white males)	Seattle & Tacoma, Wash, U.S.A.	yes, except for diseases of the arteries, veins & pulmonary circulation in FFs with 30 or more years of exposure	SMR 0.81; (0.77-0.86); n=1169	Lung cancer: SMR compared to US males: 0.96; (0.77-1.17); n=95; Trachea, bronchus and lung cancer: Incidence Density Ratio compared to police: 0.95; (0.67-1.33); no dose-response analysis for this cause	compares FFs with police, who are also screened for physical health
GUIDOTTI; (1993); standardized mortality ratio ("SMR") study	3328	1927-87 (61 yrs.)	Alberta males	Edmonton & Calgary, Alberta	no, probably because: 1-Alberta males are unusually healthy; and, 2-most subjects were traced	SMR 96.2; (86.6-106.5); n=370	Lung cancer: SMR 142; (91.0-211.4); n=24; dose-response not apparent	

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	LUNG CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
BEAUMONT et al.; (1991); mortality rate ratio ("RR") study	3066 white males	1940-82 (43 yrs.)	U.S.A. white males	San Francisco, Calif., U.S.A.	yes	RR 0.90; (0.85-0.95); n=1186	Trachea, bronchus and lung cancer: RR 0.84; (0.64-1.08); n=60; dose-response not apparent	
HEYER et al.; (1990); standardized mortality ratio ("SMR") study	2289 males	1945-83 (39 yrs.)	U.S.A. white males	Seattle, Wash., U.S.A.	yes; survivor effect also noted.	SMR 76; (69-85); n=383	Lung cancer: SMR 97; (65-139); n=29; possible dose-response	statistically significant increase in FFs aged 65 and over: SMR 177; (105-279); n=18
VENA & FIEDLER; (1987); standardized mortality ratio ("SMR") study	1867 white males	1950-79 (30 yrs.)	U.S.A. white males	Buffalo, N.Y., U.S.A.	yes, especially for circulatory system diseases.	SMR 0.95; (0.87-1.04); n=470	Respira-tory system cancer: SMR 0.94; (0.62-1.36); n=28; dose-response not apparent	
LEWIS et al.; (1982); standardized cancer mortality ratio ("SMR") study	1559	1940-80 (41 yrs.)	U.S.A. white males	Los Angeles, Calif.	yes	declining	Lung cancer: SMR for under age 70: 53.2; (39-71); n=45; 70 and over: 110.8; (80-150); n=42; no dose-response analysis	mention of any cancer on death certificate was coded as a cancer death; this may over-estimate cancer rate
TORNLING et al.; (1994); standardized mortality ratio ("SMR") study	1116	1951-86 (36 yrs.)	Stockholm males	Stockholm, Sweden	yes	SMR 82; (73-91); n=316	Lung cancer: SMR 90; (35-142); n=18; dose-response is suggested	

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	LUNG CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
DECOUFLE et al., (1977); proportionate mortality ("PMR") study	1113 stationary engineers and firefighters	1967 (1 year)	U.S. PMRs	4 areas of U.S.	not applicable	not applicable	Respira-tory system: PMR 1.22; (96-152); n=77; no dose-response analysis	combines findings for engineers and firefighters which may dilute or over-estimate results
MASTRO-MATTEO; (1959); standardized mortality ratio ("SMR") study	1039	1921-53 (33 yrs.)	Toronto & Ontario males	Toronto, Ontario	not mentioned	significant excess of deaths for all causes compared to Toronto males	no significant increase or decrease in cancer deaths; no dose-response analysis	
ELIOPULOS et al.; (1984); standardized mortality ratio ("SMR") and proportionate mortality ratio ("PMR") study	990	1939-78 (about 40 yrs.)	Western Austr. males	Western Australia	yes	SMR 0.80; (0.67-0.96); n=116	Respira-tory system cancer: SMR 0.84; (0.33 - 1.71); n=7; PMR 1.04 (0.42-2.13); no dose-response analysis	
HANSEN; (1990); standardized mortality ratio ("SMR") study	886	1970-80 (10 yrs.)	Danish civil servants	Denmark	unlikely	SMR 99; (75-129); n=57	Lung cancer: SMR 163; (75-310); n=9; no dose-response analysis	statistically significant increase in 60-74 age group (SMR 317; 117-691)
SAMA et al.; (1990); case-control study	321 FF cancer cases	1982-86 (5 yrs.)	1) 29,277 Massachusetts males; 2) 392 police	state of Massachusetts, U.S.A.	not applicable	not applicable	Lung, bronchus and trachea: Odds Ratio compared to police: 130; (84-203); compared to state: 122; (87-169); n=71; no dose-response analysis	compares FFs with general population and with police, who are also screened for physical health

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	LUNG CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
FEUER & ROSENMAN; (1986); proportional mortality ratio ("PMR") study	263 FF deaths	1974-80 (7 yrs.)	1) U.S. PMRs; 2) N.J. PMRs; 3) N.J. police PMRs	state of New Jersey, U.S.A.	not applicable	not applicable	Respiratory cancer: PMR compared to U.S. PMR: 0.98; (62-147); compared to N.J. PMR: 0.92; (58-138); compared to N.J. police PMR: 1.02; (65-153); n=23; small dose-response	compares FFs with general U.S. and state populations and with police, who are also screened for physical health
GRIMES et al.; (1991); proportional mortality ratio ("PMR") study	205 males	1969-88 (20 yrs.)	20 year and older Hawaiian males	Honolulu, Hawaii, U.S.A.	mentioned by authors	not applicable	Respiratory system: Risk Ratio: 1.28; (0.82-2.00); no dose-response analysis	rate slightly higher for Caucasians (1.09) than for Hawaiians (0.96)
MILHAM; (1983); proportional mortality ratio ("PMR") study	not stated	1950-79 (30 yrs.)	Washington state males	state of Washington, U.S.A.	not applicable	not applicable	Respiratory system: PMR 106; (85-131); n=88; no dose-response analysis	

Fig. 11


Brain Cancer Findings
Musk (103 SMR, n=8)
L'Abbe (200 SMR, n=14)
Demers (207 SMR, n=18)
Guidotti (146.6 SMR, n=3)
Vena&Fiedler (236 SMR, n=6)
Lewis (20.9 SMR, n=1) over 60
Lewis (229.3 SMR, n=13) under 60
Tornling (279 SMR, n=5)
Milham (177 PMR, N=14) CNS
Milham (180 PMR, n=13) brain
Sama (86 OR, n=5) compared to state
Sama (152 OR) compared to police
Beaumont (81 RR, n=5)
Grimes (378 RR)
Demers (163 IDR)
0 50 100 150 200 250 300 350 400
Legend
Standardized Mortality Ratio (SMR)
Proportional Mortality Ratio (PMR)
Odds Ratio (OR)
Relative Risk Ratio (RR)
Incidence Density Ratio (IDR)

(iii) Brain cancer

It is estimated that cancer of the brain accounted for 1.9% of new cancer cases and 2.6% of cancer deaths among adult males in Canada in 1993. Brain cancer incidence and mortality rates are about 30% higher among males than females [130].

More than half of all brain tumours are gliomas. "Glioma" is a term which encompasses a family of *primary* brain tumour types. It is estimated that 4% to 37% or more of brain tumours are secondary to another site [1].

All the potential causes of primary brain cancer are not yet known. Prior head injury, infection, metabolic and other systemic disease, and exposure to toxins and radiation have all been suggested as causative factors [1]. According to the Ontario Cancer Treatment and Research Foundation, occupational exposures in the rubber industry, exposure to insecticides or fungicides used in farming, to microwave and radio frequencies in the electrical industry and to vinyl chloride monomer are established causes of brain cancer [139].

Consultants' comments

Two of three consultants stated that the elevated risk of brain cancer is likely occupational in origin [111, 155]. The third said that "it is difficult to see the justification for regarding any cancer as occupational in this group." [17]

Dr. Quirt thought that there was little doubt that firefighters are at increased risk of dying from brain cancer. In addition to the finding in the IDSP study, he stated:

> " ... I am extremely impressed by the number of other studies that have pointed to an increase in brain and other central nervous system tumours in firefighters. I believe that the most valuable studies are those that compare the mortality incidence for various diseases in firefighters with those of policemen. This structure of study overcomes the problem that I mentioned previously of increasing the incidence of cancer by reducing the incidence of cardiovascular, respiratory and endocrine disease. I would suggest that the panel focus on the study conducted by Demers et al. [1992] ... This study included large numbers of firefighters (4,401) and compared them to a large cohort of police officers (3,599).
>
> . . .
>
> With several studies pointing to an increased incidence of brain cancer in firefighters and with the Demers study confirming the

increased frequency of brain tumours in firefighters when compared to policemen, I think there is little doubt that firefighters are at increased risk of dying from brain cancer.

. . .

The observation that vinyl chloride, formaldehyde, acrylonitrile, polycyclic aromatic hydrocarbons, trichloroethylene, and polychlorinated biphenyl compounds can produce brain cancer and the observations that these compounds can be detected in the inhaled substances found in the immediate environment of a fire produce a plausible reason why firefighters are at an increased risk of developing brain cancer." [155]

Dr. McDiarmid agreed, stating:

"Excesses in brain and central nervous system cancers have been seen in other working cohorts including those in rubber manufacture (Mancuso, 1982; Monson, 1976 and 1978) and polyvinyl chloride production with specific exposure to vinyl chloride monomer (Waxweiler, 1976; Cooper, 1981). Brain tumours have been induced experimentally in animals exposed to high concentrations of vinyl chloride by inhalation (Maltoni, 1977 and 1982). Others working in the oil refinery and petro-chemical production industries have demonstrated excesses as well. Alexander, 1980 demonstrated a two fold increase in risk for developing brain tumor among those employed at a Union Carbide plant in Texas and a cohort mortality study of workers employed at Union Carbide also indicated a significantly increased SMR of about two of the exposed hourly workers at the same plant (Waxweiler, 1983). Another record linkage study by Reeve, 1983 also suggested a possible increased risk at another chemical company.

Some other industry-based studies did not demonstrate an excess. The evidence would suggest however, that an excess in brain and central nervous system cancers in the workers does exist.

. . .

Several other working groups both industrial and non-industrial have demonstrated excess risk for brain cancers. These groups include embalmers, artists, pathologists and farmers (Thomas and Waxweiler, 1986). Exposure common to these groups include formaldehyde and organic solvents. The farmers' exposure opportunities may be coming from organo-chlorine pesticides or

other chemical compounds used on the farm. (Thomas and Waxweiler, 1986)

. . .

Relating these exposures to the fire environment, one can see that firefighters also share exposures to common organic substances such as formaldehyde, benzene, combustion products and vinyl chloride monomer (McDiarmid, 1991). Therefore the observation of excesses of brain and central nervous system cancer among firefighters is to be expected given that these excesses are also seen in other working cohorts who shared exposure to similar toxicants." [111]

Dr. Boyd doubted that there is an association and said:

"The question is complicated by the fact that brain neoplasms are not an homogeneous entity, but are comprised of several distinct histological types, and the risk factors that have been identified vary from one type to another.

. . .

Risk of meningiomas appears to be increased by trauma to the head, risk increasing with increasing frequency or severity of trauma. This risk would appear to be increased in fire fighters. Risk of meningiomas has also been described as increasing with exposure to passive smoking. Risk of all brain tumours and of glioblastomas has been described as increased in welders, who may have some occupational exposures in common with fire fighters.

There is one case report in the literature of an astrocytoma that may be associated with exposure to asbestos (the tumour contained an asbestos body), an exposure of possible relevance to fire fighting.

. . .

Other described risk factors for brain tumours, including radiation and exposure to electromagnetic fields, do not seem to be especially associated with fire fighting.

. . .

An initial and essential step in trying to understand the reported association between fire fighting and brain tumours is to identify the

type(s) of tumour involved. Without this information it is difficult or impossible to relate this association to other knowledge of carcinogenesis." [17]

Mortality studies

Of the 14 major studies of firefighters' mortality which gave findings for brain cancer, 11 found some excess risk. Figure 11 illustrates the findings. More detail is given in Table 12 which appears at the end of this section.

Statistically significant increases

Six mortality studies, including the IDSP study, found statistically significant increases in the rates of brain cancer among firefighters [86, 32, 121, 56, 195, 98]. Those increases occurred in the entire cohort in five of the studies, and in the most highly exposed firefighters in the sixth study [195].

The IDSP study examined the causes of 777 deaths among 5414 Toronto firefighters. It identified a statistically significant SMR of 200 (95% CI 109-336) for brain and other nervous system cancers based upon 14 deaths[16].

The study by Demers and co-workers (1992) compared 4546 firefighters with police officers. The SMR for all causes for both firefighters and police was lower than expected to a statistically significant degree, indicating a healthy worker effect for both groups[17]. Brain cancer rates, however, showed statistically significant increases among firefighters compared to US males, with an SMR of 2.07 (95% CI 1.23-3.28; n=18). An elevated rate also appeared when firefighters were compared to police (Incidence Density Ratio 1.63; [95% CI 0.70-3.79]; n=18). The increase among firefighters compared to police is particularly important because police also had a higher rate of brain cancer than expected compared to US white men[18]. No dose-response trend among firefighters was apparent in this study, however.

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16 One other case of brain cancer was excluded because the death occurred before 1950, when the study period began [86].

17 Mortality among firefighters from three northwestern U.S. cities, 1945-89:

All-cause mortality for firefighters: SMR 0.81; (95% CI 0.77-0.86); n=1169;
All-cause mortality for police: SMR 0.87; (95% CI 0.81-0.93); n=714. [32]

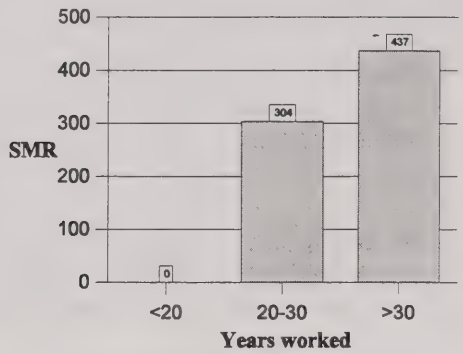
18 Mortality among police from three northwestern U.S. cities, 1945-89:

Brain cancer SMR 1.36; (95% CI 0.59-2.69); n=8.[32]

FIG. 12: BRAIN CANCER MORTALITY AMONG SWEDISH FIREFIGHTERS, 1951-1986, BY DURATION OF EMPLOYMENT AND BY NUMBER OF FIRES ATTENDED:

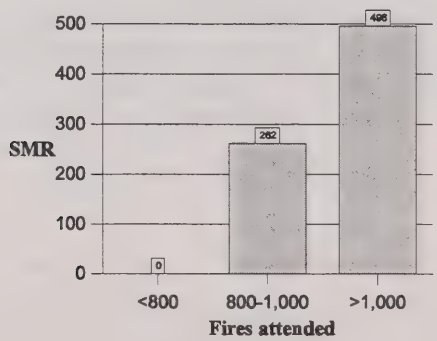
Years worked	SMR	(95%CI)	n
<20	0	(0-825)	0
20-30	304	(37-1097)	2
>30	437	(90-1278)	3

n=number of deaths



Fires attended	SMR	(95%CI)	n
<800	0	(0-611)	0
800-1,000	262	(7-1462)	1
>1,000	496	(135-1270)	4

n=number of deaths
(Tornling et al, 1990)



One of these studies, by Lewis and co-workers (1982), found an SMR of 229.3 (95% CI 122-392; n=13) for Los Angeles firefighters[19].

Tornling and colleagues (1994) found that, although the elevation in brain cancer deaths among 1116 Swedish firefighters did not reach statistical significance overall, the most highly exposed subgroup showed a statistically significant increase (Fig. 12). That study is one of only a few which provided some data on actual fire exposure, rather than duration of employment only.

The two other studies which found a statistically significant increase in brain cancer among firefighters were proportional mortality studies. Milham (1983) surveyed all occupations in Washington state from 1950 to 1979 and found a PMR of 180 ($p<0.05$), and Grimes and colleagues (1991) found a very high risk ratio of 3.78 (95% CI 1.22-11.71) among Hawaiian firefighters.

Other increases

Elevated, but not statistically significant, rates of brain cancer were also found in five other studies [202, 169, 59, 128, 43]. Some of these findings were based upon small numbers of deaths which is not surprising since cancer of the brain is relatively rare.

One of the five studies was a survey of cancer *incidence* among 321 Massachusetts firefighters between 1982 and 1986, by Sama and colleagues (1990). No increase beyond the expected number of brain cancer deaths was found in a fairly short time frame. When firefighters were compared with police, however, the rate was higher but did not reach statistical significance (OR 152; 95% CI 39-392; n=5).

Discussion

The following factors may have had some impact on the epidemiological data.

Appropriateness of study period

The Panel examined the findings of the mortality studies to determine which of them covered a sufficiently long time period following the widespread introduction of plastics in the mid 1950s to capture an excess in mortality from brain cancer. There were eleven mortality studies which found some elevation

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[19] Heyer (1988), however, thought that figure could be inflated because any death certificate which mentioned any cancer was classified as a cancer death, whether or not cancer was the primary cause or the only potentially fatal condition. But since cancers of the brain are usually rapidly fatal [1] and would probably cause death sooner than other co-existing conditions which are potentially fatal, the findings for cancer of the brain may be less inaccurate than findings for other cancers.

in the rate of brain cancer, and their average duration of follow-up from 1955 onward was about 25 years. The studies which did not identify that association, however, provided an average duration of follow-up after 1955 of only 13.75 years (eg. Mastromatteo's 1959 study spanned the years 1918 to 1954).

Those studies which did not identify an association probably did not cover the appropriate study period following the widespread introduction of plastics in the 1950's.

Primary vs. secondary tumours

Because the brain is commonly a site for secondary cancers to develop, it may be that a brain cancer shown as the cause of death on a death certificate was not the primary cancer site. The Panel, therefore, attempted to examine the types of tumours in order to determine whether the brain cancers were primary or secondary.

Prior to 1969, the internationally-used method for classifying causes of death (International Classification of Diseases, or "ICD codes") did not specifically identify tumour types for brain cancer. As a result, mortality studies which have had to rely upon ICD codes to identify causes of death were not able to specify the type of tumour for deaths that occurred before 1969. As a consequence, we cannot always know whether the cases of brain cancer before that time were primary tumours or were secondary to some other site of cancer.

In the IDSP study, six of the fourteen brain cancers were confirmed by the Ontario Cancer Treatment and Research Foundation as gliomas [86], that is, they were *primary* brain tumours.

Only three of the other studies had information about tumour type in some of their cases [32, 195, 43]. The Demers (1992) cohort had a statistically significant excess of brain cancer mortality of which thirteen were gliomas and nine were brain tumours of unspecified type.

All of the five brain cancer deaths reported by Tornling and co-workers (1994) were due to gliomas.

Finally, the Australian cohort described by Eliopulos and colleagues (1984) included two firefighters who died of gliomas, but their deaths occurred within the first year of starting work. Given that latency periods for cancer are thought to be five years or longer [111], these two deaths were highly unlikely to be work-related.

Since the other studies do not specify the types of brain tumours which occurred in either the study populations or the comparison groups, it is not possible to know whether they were primary or secondary tumours. Some authors, however, assume that the proportion of primary brain tumours among workers is about the same as that which occurs in the general population [191].

If chemical exposures increase the risk of primary brain cancer, the proportion of brain cancers that are primary tumours would be higher in an exposed group of workers than in the general population. If chemical exposures are *not* a cause of brain tumours, there is no reason to suspect that there would be fewer primary brain tumours in a working population than in the general population -- there would be at least the same proportion of primary brain tumours in the working population.

Diagnostic sensitivity bias

There is evidence that some cohorts of workers receive more complete medical evaluations than controls and appear to experience higher rates of brain cancer. An apparent increase identified in Eastman Kodak workers was thought to be attributable to more thorough diagnostic studies of those workers compared to the general population [54]. The effect of this is known as *diagnostic sensitivity bias*.

It is not likely, however, that bias accounted for the statistically significant brain cancer excesses found in widely varying types of white and blue-collar occupations, largely in the US, by Thomas and Waxweiler (1986). The authors state that such a bias might

> " ... appear plausible in light of the excess risk noted in several professional and white-collar groups; however, all of the workers reported to have elevated brain tumor risk in this review were probably not enrolled in comprehensive medical surveillance programs. Furthermore, excess brain tumours were seen only among certain subgroups of some plant populations (eg. workers in the tire building sector of rubber production, refinery workers in lubricating oil manufacture). In addition, other groups with medical surveillance programs, for example, DuPont employees, do not show an elevated brain tumor risk." [191]

This potential source of bias becomes even less important in Canada where universal health care has existed for decades.

Multiple tests of significance

This refers to the possibility that when numerous disease outcomes are examined, statistically significant associations will be found for some of them by chance alone.

The IDSP mortality study reported findings for numerous causes of death among Toronto firefighters, including a statistically significant excess due to brain cancer. The Panel asked one of the study's authors, George Tomlinson, to investigate this potential source of bias. By the Schweder and Spjotvoll (1982) method, Mr. Tomlinson determined that the brain cancer SMR of 200 has a notably small p-value of 0.009, meaning that there is less than a 0.9% possibility that this excess resulted from multiple tests of significance [193].

After giving careful consideration to each of these potential sources of bias, the Panel concluded that neither separately nor jointly did these issues bias the findings. Therefore, the findings appear to accurately reflect the rates of brain cancer among firefighters.

Dose-response analyses

Only five of the fourteen studies mentioned above provided results by duration of employment which is necessary in order to conduct a dose-response analysis. One of them identified a dose-response trend [195] but the other four did not [86, 32, 68, 202].

As discussed above, Tornling and colleagues (1994) provided analyses both by number of fires attended and by duration of employment, rather than by duration of employment only. Both analyses indicated a dose-response trend, which was stronger for number of fires attended than for duration of employment. The group which attended 1000 or more fires experienced a statistically significant increase in brain cancer deaths (Fig. 12).

Potential causative agents

Vinyl chloride

According to IARC, there is sufficient evidence (Group 1) to establish that vinyl chloride causes cancer of the brain in humans [79]. Studies have shown an association between vinyl chloride and brain tumours, particularly gliomas [191, 93]. Gliomas have been induced in experimental animals by inhalation of vinyl chloride and acrylonitrile [191], both of which are designated substances in Ontario [141, 145].

Vinyl chloride is used primarily to manufacture plastic articles (from polyvinyl chloride) such as building and construction materials, packaging, clothing, insulation, automobile upholstery and a variety of consumer goods [21]. These plastic articles are commonly present in building and material fires.

When sufficiently heated, these plastic articles undergo thermal decomposition and can release vinyl chloride vapour. Small amounts of vinyl chloride have been measured in the smoke of burning plastics in fires [106, 40].

Acrylonitrile

IARC describes acrylonitrile as probably carcinogenic (Group 2A) in the case of brain cancer [79]. This substance is widely used in textile fibres and rubber, materials that are made into clothing, building materials and household products [164].

None of the researchers tested for the presence or amount of acrylonitrile in their chemical analysis of fire smoke. The IDSP's Industrial Hygienist advised the Panel that thermal decomposition of various rubber products and textile materials would result in the preserve of acrylonitrile vapours at fire sites.

Formaldehyde

IARC reports that formaldehyde is probably carcinogenic to humans (Group 2A). Excess mortality due to brain cancer was reported in more than one study [79].

Formaldehyde is used in the manufacture of resins, textiles, embalming fluids, fungicides, air fresheners [20], plastics, adhesives, wood products, insulation, paints, leather and rubber [164]. It may therefore be present as a decomposition product in fires involving such materials. Levels which could cause acute health effects in humans have been measured in studies of different types of fires [19, 82].

Other information

The authors of a review of the literature on firefighters and cancer conducted for the British Columbia Workers' Compensation Board concluded that

" ... there appear to be positive associations with brain tumours, malignant melanoma and multiple myeloma, with the evidence in favour of causality being somewhat greater for brain tumours and multiple myeloma, than for malignant melanoma." [74]

Summary of the evidence

- A strong association was shown in six studies that found statistically significant excesses of brain cancer among firefighters, with SMRs of 200, 207, 229.3 and 496, a PMR of 180 and an RR of 3.78.

- Consistency of associated is demonstrated by five other studies that found elevated rates which did not reach statistical significance.

- IARC reports sufficient evidence to establish an association between brain cancer and vinyl chloride exposure. In addition, formaldehyde and acrylonitrile are probable brain carcinogens. These are substances which would commonly be present at fire sites.

- The firefighter mortality studies which did not identify an excess probably did not cover the appropriate study period after the widespread introduction of plastics in the mid-1950's.

- The only study which estimated actual fire exposure in its dose-response analyses found a clear dose-response trend. The four other studies that conducted dose-response analyses did not identify such a trend but used duration of employment as a surrogate for exposure. Duration of employment does not provide meaningful information about "dose", hence the lack of a dose-response trend in these studies does not invalidate a causal relationship.

- Two of the three IDSP medical consultants reported that an association with firefighting is biologically plausible and coherent with other scientific knowledge about brain cancer because:

  - statistically significant excesses in brain cancer mortality have also been identified among workers in other occupations, some of whose common exposures are also known to be present at fire sites (such as vinyl chloride, formaldehyde, acrylonitrile and PAHs); and

  - gliomas have been experimentally induced in animals by inhalation of acrylonitrile and vinyl chloride, the same route of entry involved in firefighters' exposure.

- The third consultant reported that it is necessary to know the tumour type before a potential association can be identified. Although a substantial proportion of the brain tumour types could not be identified, where tumour type was identified they were all (primary) gliomas. For example, in the Tornling et al. study (1994), which found a nearly significant excess of brain cancer deaths, all five of the tumours were gliomas.

The Panel's conclusions and finding

Applying the criteria suggested by Bradford Hill, the Panel has accepted that a strong statistical association is shown between firefighting and primary brain cancer. That association has been repeatedly and consistently identified in a number of firefighter studies. The study that measured actual fire exposure indicated a dose-response trend. Substances have been identified at fire sites which are recognized brain carcinogens.

Accordingly, the Panel makes the following finding.

- **A probable connection exists between firefighting and primary cancer of the brain.**

Because of the evidence of a probable connection between firefighting and primary cancer of the brain, the Panel concludes that the disease and the process should be included in a Schedule appended to the Regulations to the *Workers' Compensation Act*. The occurrence of primary brain cancer in the general population and the lack of more specific knowledge about its causes have persuaded the Panel that the entry should be included in Schedule 3, but not Schedule 4.

This recommendation would give a firefighter a presumption that the brain cancer was due to employment, unless the contrary is proved. This would mean that such a firefighter would receive workers' compensation benefits unless it could be proven that the brain cancer was not work related. Evidence which might prove the contrary could include information about latency, alternative causes or duration of employment as a firefighter. To allow the WCB adjudicators to fairly and equitably assess the evidence that would be used to rebut the presumption, it is critical to develop guidelines or a rebuttal matrix.

The Panel's recommendations

- **"Primary cancer of the brain" and the associated process, trade or occupation of "firefighter" should be added to Schedule 3 of the *Act*.**

- **A rebuttal matrix approved by the Panel should be used to assess the evidence used to rebut the presumption.**

TABLE 12: FIREFIGHTER MORTALITY/MORBIDITY STUDIES: BRAIN CANCER FINDINGS

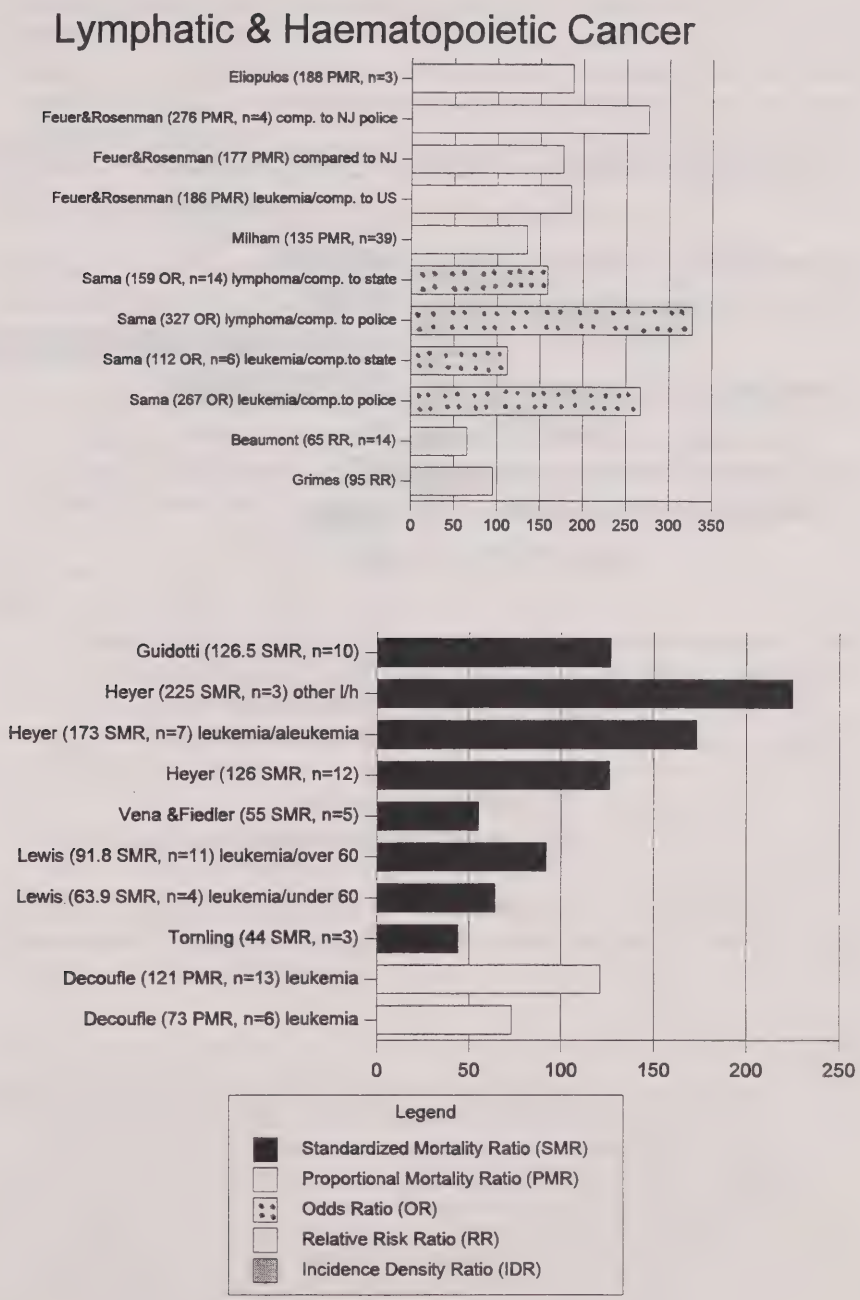
AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	BRAIN CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
MUSK et al.; (1978); standardized mortality ratio ("SMR") study	5655 males	1915-75 (61 yrs.)	male population of Mass. and U.S.A.	Boston, Mass., U.S.A.	yes	2470 deaths; 91% of expected for Mass. males; 94% of expected for U.S.A. white males	Brain & Other CNS: SMR 103; (44-203); n=8; no dose-response analysis	
L'ABBÉ & TOMLINSON; (1992); (IDSP study) standardized mortality ratio ("SMR") study	5,414 males; 5,373 for duration of employment analysis	1950-89 (40 yrs.)	Ontario males	Metropolitan Toronto	strong for FFs under 40 and for FFs with less than 10 years since first exposure	SMR 94; (88-101); n=777	Brain & Other CNS: SMR 200; (109-336); n=14; (6 gliomas; 8 unspeci-fied); dose-response not apparent	
DEMERS et al.; (1992); standardized mortality ratio ("SMR") study	4546 males	1945-89 (45 yrs.)	1) U.S. white males; 2) 3676 police from Seattle & Tacoma, Wash., U.S.A. (white males)	Seattle & Tacoma, Wash, U.S.A.	yes, except for diseases of the arteries, veins & pulmonary circulation in FFs with 30 or more years of exposure	SMR 0.81; (0.77-0.86); n=1169	Brain and nervous system cancers: compared to U.S. males: SMR 2.07; (1.23-3.38); n=18; (13 gliomas; 5 unspeci-fied malig-nant); compared to police: Incidence Density Ratio: 1.63; (0.79-3.79); dose-response not apparent	compares FFs with police, who are also screened for physical health

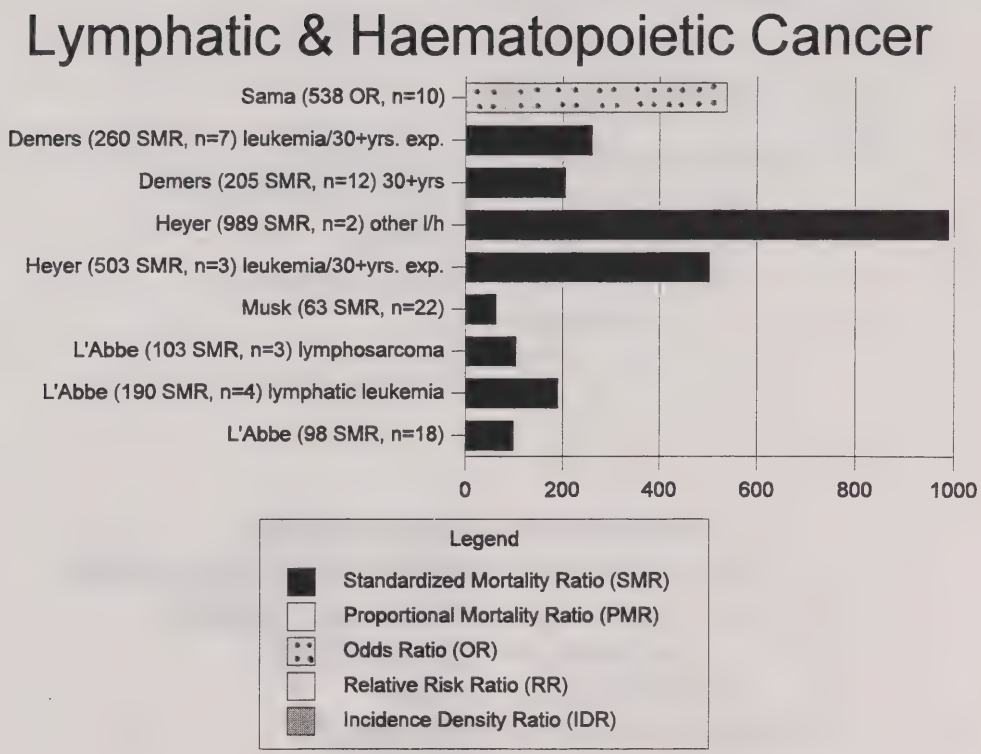
AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	BRAIN CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
GUIDOTTI; (1993); standardized mortality ratio ("SMR") study	3328	1927-87 (61 yrs.)	Alberta males	Edmonton & Calgary, Alberta	no, probably because: 1-Alberta males are unusually healthy; and, 2-most subjects were traced	SMR 96.2; (86.6-106.5); n=370	Brain Cancer: SMR 146.6; (30.3-428.5); n=3; no dose-response analysis for this cause	
BEAUMONT et al.; (1991); mortality rate ratio ("RR") study	3066 white males	1940-82 (43 yrs.)	U.S.A. white males	San Francisco, Calif., U.S.A.	yes	RR 0.90; (0.85-0.95); n=1186	Brain Cancer: RR 0.81; (0.26-1.90); n=5; no dose-response analysis for this cause	
HEYER et al.; (1990); standardized mortality ratio ("SMR") study	2289 males	1945-83 (39 yrs.)	U.S.A. white males	Seattle, Wash., U.S.A.	yes; survivor effect also noted.	SMR 76; (69-85); n=383	Brain Cancer: elevated during first 15 years since first exposure, but less than expected thereafter; no dose-response shown	
VENA & FIEDLER; (1987); standardized mortality ratio ("SMR") study	1867 white males	1950-79 (30 yrs.)	U.S.A. white males	Buffalo, N.Y., U.S.A.	yes, especially for circulatory system diseases.	SMR 0.95; (0.87-1.04); n=470	Brain & Other CNS: SMR 2.36; (0.86-5.13); n=6; dose-response not apparent	
LEWIS et al.; (1982); standardized cancer mortality ratio ("SMR") study	1559	1940-80 (41 yrs.)	U.S.A. white males	Los Angeles, Calif.	yes	declining	Brain cancer: SMR for under age 60: 229.3; (122-392); n=13; 60 and over: 20.9; (1-116); n=1; no dose-response analysis	mention of any cancer on death certifi-cate was coded as a cancer death; this may over-estimate cancer rate

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	BRAIN CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
TORNLING et al.; (1994); standardized mortality ratio ("SMR") study	1116	1951-86 (36 yrs.)	Stockholm males	Stockholm, Sweden	yes	SMR 82; (73-91); n=316	Brain tumours, glioma: SMR 279; (91-651); n=5; dose-response is shown	SMR for those who attended 1000 or more fires: 496; (135-1270); n=3
MASTRO-MATTEO; (1959); standardized mortality ratio ("SMR") study	1039	1921-53 (33 yrs.)	Toronto & Ontario males	Toronto, Ontario	not mentioned	significant excess of deaths for all causes compared to Toronto males	no significant increase or decrease in cancer deaths; no dose-response analysis	
ELIOPULOS et al.; (1984); standardized mortality ratio ("SMR") and proportionate mortality ratio ("PMR") study	990	1939-78 (about 40 yrs.)	Western Australia males	Western Australia	yes	SMR 0.80; (0.67-0.96); n=116	Brain: SMR 8.333; (1.009-30.103) n=2; both gliomas; both deaths occurred within 1 year of hire; no dose-response analysis	
SAMA et al.; (1990); case-control study	321 FF cancer cases	1982-86 (5 yrs.)	1) 29,277 Massachusetts males; 2) 392 police	state of Massachusetts, U.S.A.	not applicable	not applicable	Brain & other CNS: Odds Ratio compared to police: 152; (39-592); compared to state: 86; (34-215); n=5; no dose-response analysis	compares FFs with general popula-tion and with police, who are also screened for physical health
GRIMES et al.; (1991); proportional mortality ratio ("PMR") study	205 males	1969-88 (20 yrs.)	20 year and older Hawaiian males	Honolulu, Hawaii, U.S.A.	yes	not applicable	Brain & other CNS: Risk Ratio 3.78; (1.22-11.71); no dose-response analysis	

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	BRAIN CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
MILHAM; (1983); proportional mortality ratio ("PMR") study	not stated	1950-79 (30 yrs.)	Washington state males	state of Washington, U.S.A.	not applicable	not applicable	Brain & CNS: PMR 177; ($p<0.05$); n=14; Brain: PMR 180; ($p<0.05$); n=13; no dose-response analysis	

Fig. 13


Lymphatic & Haematopoietic Cancer
Eliopulos (188 PMR, n=3)
Feuer&Rosenman (276 PMR, n=4) comp. to NJ police
Feuer&Rosenman (177 PMR) compared to NJ
Feuer&Rosenman (186 PMR) leukemia/comp. to US
Milham (135 PMR, n=39)
Sama (159 OR, n=14) lymphoma/comp. to state
Sama (327 OR) lymphoma/comp. to police
Sama (112 OR, n=6) leukemia/comp.to state
Sama (267 OR) leukemia/comp.to police
Beaumont (65 RR, n=14)
Grimes (95 RR)
0 50 100 150 200 250 300 350
Guidotti (126.5 SMR, n=10)
Heyer (225 SMR, n=3) other l/h
Heyer (173 SMR, n=7) leukemia/aleukemia
Heyer (126 SMR, n=12)
Vena &Fiedler (55 SMR, n=5)
Lewis (91.8 SMR, n=11) leukemia/over 60
Lewis (63.9 SMR, n=4) leukemia/under 60
Tornling (44 SMR, n=3)
Decoufle (121 PMR, n=13) leukemia
Decoufle (73 PMR, n=6) leukemia
0 50 100 150 200 250
Legend
Standardized Mortality Ratio (SMR)
Proportional Mortality Ratio (PMR)
Odds Ratio (OR)
Relative Risk Ratio (RR)
Incidence Density Ratio (IDR)


Lymphatic & Haematopoietic Cancer
Sama (538 OR, n=10)
Demers (260 SMR, n=7) leukemia/30+yrs. exp.
Demers (205 SMR, n=12) 30+yrs
Heyer (989 SMR, n=2) other l/h
Heyer (503 SMR, n=3) leukemia/30+yrs. exp.
Musk (63 SMR, n=22)
L'Abbe (103 SMR, n=3) lymphosarcoma
L'Abbe (190 SMR, n=4) lymphatic leukemia
L'Abbe (98 SMR, n=18)
0
200
400
600
800
1000
Legend
Standardized Mortality Ratio (SMR)
Proportional Mortality Ratio (PMR)
Odds Ratio (OR)
Relative Risk Ratio (RR)
Incidence Density Ratio (IDR)

(iv) Lymphatic and haematopoietic cancers

This category includes cancers arising in the family of cells responsible for the blood, the immune system and host defence[20]. Many of the studies referred to below gave findings for the broad category of lymphatic and haematopoietic cancers combined, while some also provided separate rates for specific cancers included within that category, such as leukaemia or lymphoma. The varying categories in which these findings were originally reported by the authors made comparisons difficult. It was not possible to re-organize the data into a more comparable format.

Consultants' comments

Only one of the Panel's three consultant carcinogenecists commented on a potential link between these types of cancer and occupational exposures. Dr. McDiarmid noted that

> "Certain types of leukaemia have been noted in excess in various other working cohorts including those exposed to benzene (Vigilani, 1976; Infante, 1986; Rinsky, [1987]) and agricultural workers. Benzene has been found in several exposure assessments of the firefighting environment.
>
> . . .
>
> "Excesses of lymphoma have been seen in some pesticide workers. In this study numbers are extremely small. One view is that lymphoma should be viewed as a solid tumor form of leukaemia and thus certain exposures known to cause leukaemia may be implicated in the cause of lymphoma." [111]

Mortality and morbidity studies

Figure 13 illustrates the findings of seventeen studies of lymphatic and haematopoietic cancers in firefighters. More detail is given in Table 13 which appears at the end of this section.

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20 "Lymphatic and haematopoietic tissue neoplasms" comprise neoplasms arising from the families of blood-borne cells and those in various other tissues which share a common origination within a pool of less differentiated "stem cells". The haematopoietic stem cell system consists of pools of proliferating cells of various degrees of differentiation leading to the "mature" families of the white cells (lymphocytes, platelets) and those found in tissues (spleen, liver, lymph nodes, bone marrow, etc.) such as macrophages and plasma cells engaged in immune response and host defence (the reticuloendothelial system). As neoplasms, they manifest diversely as populations of abnormal white cells in the blood (leukaemia), solid tumours in lymph nodes or elsewhere (lymphoma) with a wide range of rates of progression and lethality [23].

Within these seventeen firefighter studies, six statistically significant increases in lymphatic/haematopoietic cancer deaths were reported. Two of these were found for the overall cohort, and four occurred in subsets. Eleven elevated rates which did not reach statistical significance were also reported. Six studies found no increase or non-statistically significant decreases in these cancers. One author reported a statistically significant decrease.

Four analyses were suggestive of a dose-response trend in some categories, two did not show a dose-response and the other studies did not conduct dose-response analyses.

Another study, of cancer mortality among various occupations, found a statistically significant increase in lymphatic/haematopoietic cancer deaths among Massachusetts firefighters.

Statistically significant increases in overall cohort

Non-Hodgkin's lymphoma was elevated to a statistically significant degree among Massachusetts firefighters between 1982 and 1986 compared to that among police officers (Odds Ratio 327; 95% CI 119-898) [169]. This study examined the incidence of cancer based on state Cancer Registry records and therefore it did not encounter the inaccuracies known to occur in death certificate data [172]. The authors thought that the findings could underestimate the true incidence of firefighters' cancer because occupational information was available for only about 50% of all Massachusetts Cancer Registry cases.

A statistically significant increase in leukaemia among 263 New Jersey firefighter deaths was identified by Feuer and Rosenman (1986) compared with police (SMR 276; $p<0.05$).

Statistically significant increases in subsets

Among 4546 northwestern US firefighters studied by Demers et al. (1992), the broader category of lymphatic/haematopoietic cancer was elevated in the cohort as a whole, but not to a statistically significant degree. The increase was statistically significant for those with 30 or more years employment (SMR 205; 95% CI 110-360). Leukaemia specifically was increased to a statistically significant degree for firefighters with 30 or more years of service, with an SMR of 260 (95% CI 100-540).

Similarly, Heyer and colleagues (1990) report that the SMRs for all lymphatic/haematopoietic cancers among 2289 Seattle firefighters were elevated but did not reach statistical significance when the group was

considered as a whole; however, for firefighters with 30 or more years of service, the increases in both leukaemia (SMR 503; 95% CI 104-1470) and other lymphatic/haematopoietic cancers (SMR 989; 95% CI 120-3571) reached statistical significance.

Other findings

Six sets of authors identified increased rates of some types of lymphatic/haematopoietic cancers which did not reach statistical significance [86, 121, 43, 59, 169, 31].

Six others found no increase or decreases which were not statistically significant [202, 10, 195, 56, 98,109]. Musk et al. (1978) identified a statistically significant decrease in these cancers, with an SMR of 63 (95% CI 39-95; n=22).

Finally, the published report on the largest firefighter mortality study, by Orris and co-workers, involved 3084 deaths among Chicago firefighters but did not specify results for leukaemia. The Panel asked Dr. Orris' colleague, Dr. Paul Targonski, to re-examine their data and isolate findings for several types of cancer which were elevated in some of the other studies [190]. The data have been updated and now include 3314 deaths. The overall leukaemia rate for the period 1948-1982 was not remarkable[21].

Discussion

The Panel examined the findings of the mortality studies discussed above to determine which of them covered a sufficiently long period following the widespread use of plastics since the mid 1950's to capture the mortality from lymphatic and haematopoietic cancers which might result from such exposures. Excluding the Decoufle study (1977), which is uninformative because it combined firefighters with stationary engineers, there were nine mortality studies which found some elevation in rates of these cancers The average duration of follow-up from 1955 onward was 25.22 years, ranging from 0 to 28 years.

The seven studies which did not identify that association provided an average duration of follow-up of 20 years since 1955 (ranging from 0 to 32 years). The studies with negative results provide an only slightly shorter follow-up period after the introduction of plastic materials.

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[21] Dr. Targonski reported only one statistically significant increase in leukaemia deaths, which occurred during the short period between 1978 to 1982 (PMR 2.382; $p<0.005$; 95% CI 1.191-4.224; n=11) [190].

FIG. 14: LYMPHATIC/HAEMATOPOIETIC CANCERS OTHER THAN LEUKEMIA AMONG SEATTLE FIREFIGHTERS WITH 30 OR MORE YEARS OF SERVICE, 1945-1983, BY DURATION OF EXPOSURE:

Years worked	SMR	(95%CI)	n
<15	0	(0-4,445)	0
15-29	160	(4-892)	1
30+	989	(120-3,571)	2

n=number of deaths
(Heyer et al., 1990)

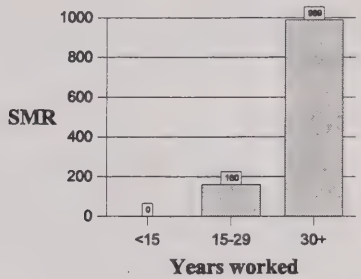


FIG. 15: CANCER OF BLOOD AND BLOOD CELLS MORTALITY AMONG SWEDISH FIREFIGHTERS, 1951-1986, BY NUMBER OF FIRES ATTENDED:

Fires attended	SMR	(95%CI)	n
under 800	0	(0-194)	0
800-1,000	54	(1-302)	1
1,000+	64	(8-230)	2

n=number of deaths
(Tornling et al., 1990)

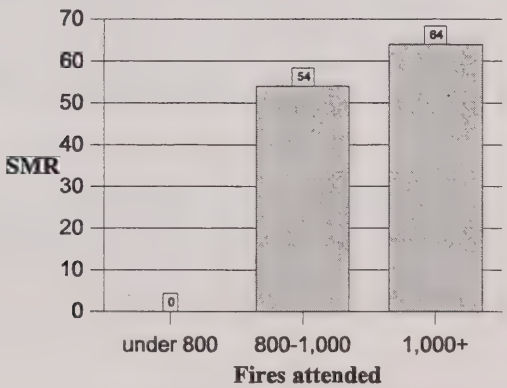


FIG. 16: LYMPHATIC HAEMATOPOIETIC CANCER MORTALITY AMONG FIREFIGHTERS FROM THREE NORTHWESTERN U.S. CITIES, 1945-85, BY DURATION OF EMPLOYMENT:

Years worked	SMR	(95%CI)	n
<10	0.91	(0.2-2.3)	4
10-19	1.46	(0.06-3.0)	7
20-29	1.06	(0.06-1.8)	14
30+	2.05	(1.1-3.6)	12

n=number of deaths
(Demers et al., 1992)

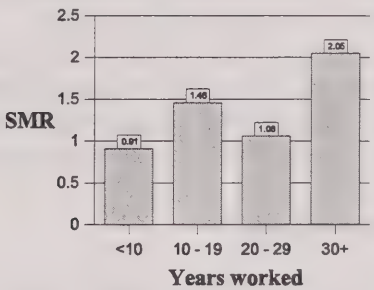
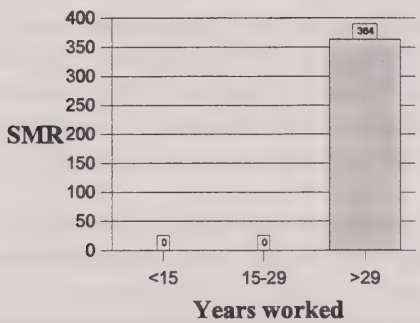


FIG. 17: LYMPHATIC LEUKAEMIA MORTALITY AMONG METROPOLITAN TORONTO FIREFIGHTERS, 1950-1989, BY DURATION OF EMPLOYMENT:

Years worked	SMR	(95%CI)	n
<15	0.00		0
15-29	0.00		0
>29	364.00	(99-932)	4

n=number of deaths
(L'Abbe and Tomlinson., 1992)
(IDSP study)



Dose-response analyses

Lymphatic/haematopoietic cancers other than leukaemia among 2289 Seattle firefighters increased with duration of employment suggesting a dose-response trend (Fig. 14) [68].

Although Tornling and colleagues (1994) identified a dose-response trend, the overall SMR was only 44 and the SMR for those with the greatest exposure was only 64 (Fig. 15). That study is one of only two that provided some data on actual fire exposure, rather than duration of employment only.

Although the rates did not increase consistently with duration of employment in either category, a weak dose-response trend may have occurred among northwestern US firefighters (Fig.16) [32].

While the SMRs for lymphatic leukaemia did not reach statistical significance in the IDSP study, all four deaths occurred in the group with 30 or more years of employment. This may indicate a dose-response (Fig. 17) [86].

Potential causative agents

Benzene

IARC reports sufficient evidence (Group 1) that benzene and soots are carcinogenic to humans, citing several studies linking benzene with leukaemia as well as one that implicated soots (which contain PAHs) which are present at every fire [79].

After carbon monoxide, benzene is generally accepted to be the second most commonly found organic constituent of fire smoke, typically present in high concentrations in the fire environment. Benzene is used as an ingredient for the manufacture of various products (e.g. medicinal chemicals, dyes, artificial leather, linoleum, oil cloth, varnishes and lacquers) and as a solvent for waxes, resins and oils [20]. It is also a common decomposition product of many organic materials and diesel exhaust. Significant levels have been measured at various content/building and car fires [19, 198].

A more recent study showed that firefighters could be highly exposed to benzene even with the use of self-contained breathing apparatus. Levels as high as 21 ppm were measured from air samples collected from *inside* SCBA masks worn at fire sites [82]. The current maximum allowable concentration of benzene in Ontario workplaces is 15 ppm [144].

Lymphoid cancer has been induced in mice by inhalation of benzene [79].

Soots and PAHs

IARC notes statistically significant excesses in mortality from scrotal and other cancers among chimney sweeps exposed to soots, a Group 1 carcinogen [79]. A statistically significant excess in leukaemia mortality was also found in one study. Soots contain PAHs, many of which are known carcinogens to humans.

Leukaemia has been linked to exposure to PAHs [62], which are multi-ring aromatic compounds found widely dispersed in nature. They are formed during the combustion of many organic materials as well as in tobacco smoke and grilled, smoked and fried foods [81].

It is likely that firefighters are significantly exposed to soots during firefighting since all fires generate soots. Only one study reported measured airborne PAHs in fire smoke. The measurements ranged from below detection to 0.5 mg/m^3 during knockdown and below detection to 0.02 mg/m^3 during overhaul [82].

Vinyl chloride

According to IARC, there is sufficient evidence (Group 1) to establish an association between vinyl chloride exposure and haematolymphopoietic cancers in humans [79].

As discussed in the case of brain cancer, vinyl chloride is used primarily to manufacture plastic articles (from polyvinyl chloride) such as building and construction materials, packaging, clothing, insulation, automobile upholstery and a variety of consumer goods [60]. These plastic articles are commonly present in building and material fires.

When sufficiently heated, these plastic articles undergo thermal decomposition and can release vinyl chloride vapour. Small amounts of vinyl chloride have been measured in the smoke of burning plastics in fires [106].

Acrylonitrile

IARC reports that acrylonitrile is a probable carcinogen (Group 2A) causing lymphatic and haematopoietic cancers [79].

Acrylonitrile is widely used in textile and rubber materials that are made into building materials and household goods. None of the studies reviewed included acrylonitrile in their chemical analysis of fire smoke. However, the Panel's Industrial Hygienist advises that firefighters could be frequently exposed to

acrylonitrile vapours generated from thermal decomposition of various rubber products and textile materials.

Formaldehyde

IARC describes formaldehyde as probably carcinogenic (Group 2A) and cites evidence that it can cause Hodgkin's disease and leukaemia [79].

Formaldehyde is used in the manufacture of resins, textiles, embalming fluids, fungicides, air fresheners [20], plastics, adhesives, wood products, insulation, paints, leather and rubber [164]. It may therefore be present as a decomposition product in fires involving such materials. Levels which could cause acute health effects in humans have been measured in studies of different types of fires [19, 82].

Because of their recognized danger, benzene, vinyl chloride and acrylonitrile are "designated substances" in Ontario [144, 141, 145].

Other information

Dubrow and Wegman [35] studied cancer mortality patterns by occupation for white males in Massachusetts using 1971-1973 death records. In order to organize this large amount of data for their published report, the authors reported their findings by occupational categories which were as homogeneous as possible, so as not to dilute or cancel out possible effects. At the same time, they created grouped codes of occupations that were likely to have similar exposures. Since there were so few firefighter deaths, the occupation of firefighter was not included in the published report. The scientific report of that study, however, showed a statistically significant excess of deaths from cancers of the lymphatic and haematopoietic tissue among Massachusetts firefighters aged 20 to 54[22] [36].

[22] Occupational characteristics of cancer victims in Massachusetts, 1971-1973:

Lymphatic and haematopoietic tissue: Odds Ratio 241; $p<0.05$; n=6. [36]

Summary of the evidence

- A strong association was identified in six studies. SMRs ranged from 205 to 989. While four of those findings were for subgroups of long-serving firefighters, two of them were for entire cohorts compared to another group of workers, police officers, who are also screened for physical health before being hired. Comparison with a working population is a method recommended for controlling for the healthy worker effect. In addition, one of the studies which found a statistically significant increase in lymphatic/haematopoietic cancers among firefighters studied cancer incidence, a study design preferred over mortality studies by one of the Panel's expert consultants when investigating these types of cancer.

- Four analyses identified a dose-response trend but two did not. As discussed above, when the "dose" experienced by firefighters is unknown, it is impossible to generate a dose-response trend. In addition, when studying relatively rare diseases, the number of deaths will be small. The lack of a dose-response trend when the numbers are small is evidence neither for nor against an association. Analyses involving small numbers which do identify a dose-response trend, however, support an association. The findings of statistically significant increases in long-serving firefighters also provides evidence suggestive of a dose-response.

- While firefighters' occupational exposures have not so far been well characterized, there is evidence that benzene is the second most prevalent exposure at fire sites. Significant levels of benzene have been measured in air samples taken from inside self-contained breathing apparatus masks worn at fire sites. Benzene is recognized by IARC as a Group 1 carcinogen for which there is sufficient evidence of a causal relationship with various types of leukaemia.

- The induction of lymphoid cancer in animal experiments by inhalation of benzene, the same route of entry experienced by firefighters, provides further evidence in support of an association.

- It can be assumed that soots are commonly present at fire sites. IARC describes soots as a Group 1 carcinogen associated with several types of cancer and with leukaemia in one study.

- While the level of vinyl chloride in fire smoke was low in the only study which measured it, vinyl chloride is a component of plastics used in pipes, ducts, floor tiles, electrical wire, packaging, clothing, insulation, upholstery and a variety of consumer goods. Vinyl chloride is released when these common materials are combusted and it is therefore likely to be present at most fires. IARC describes vinyl chloride as a Group 1 carcinogen for

which there is sufficient evidence of an association with haematolymphopoietic cancers in humans.

The Panel's conclusions and finding

Although increases did not appear consistently throughout the mortality and morbidity data, several studies found a strong statistical association. The statistically significant excesses among long-serving firefighters provides additional support for the evidence of a dose-response in some studies. A connection between these cancers and working as a firefighter is biologically plausible given their established occupational exposure to known lymphatic/haematopoietic carcinogens. Finally, the induction of these cancers by inhalation of benzene, a prevalent exposure at fire sites, provides experimental evidence in support of a probable connection.

Accordingly, the Panel makes the following finding.

- **A probable connection exists between firefighting and primary lymphatic and haematopoietic cancers.**

Because of the weight of evidence in support of a probable connection between firefighting and these types of cancers, the Panel concludes that the disease and the process should be included in a Schedule appended to the Regulations to the *Workers' Compensation Act*. The fact that primary lymphatic and haematopoietic cancers occur in the general population due to non-occupational causes has persuaded the Panel that the entry should be included in Schedule 3, but not Schedule 4.

This recommendation would give a firefighter a presumption that the lymphatic and haematopoietic cancer was due to employment, unless the contrary is proved. This would mean that such a firefighter would receive workers' compensation benefits unless it could be proven that the cancer was not related to the firefighting. Evidence which might prove the contrary could include information about latency or duration of employment as a firefighter. To allow the WCB adjudicators to fairly and equitably assess the evidence that would be used to rebut the presumption, it is necessary to develop guidelines or a rebuttal matrix.

The Panel's recommendations

- **"Primary lymphatic or haematopoietic cancer" and the associated process, trade or occupation of "firefighter" should be added to Schedule 3 of the *Act*.**

- **A rebuttal matrix approved by the Panel should be used to assess the evidence used to rebut the presumption.**

TABLE 13: FIREFIGHTER MORTALITY/MORBIDITY STUDIES: LYMPHATIC/HAEMATOPOETIC CANCERS

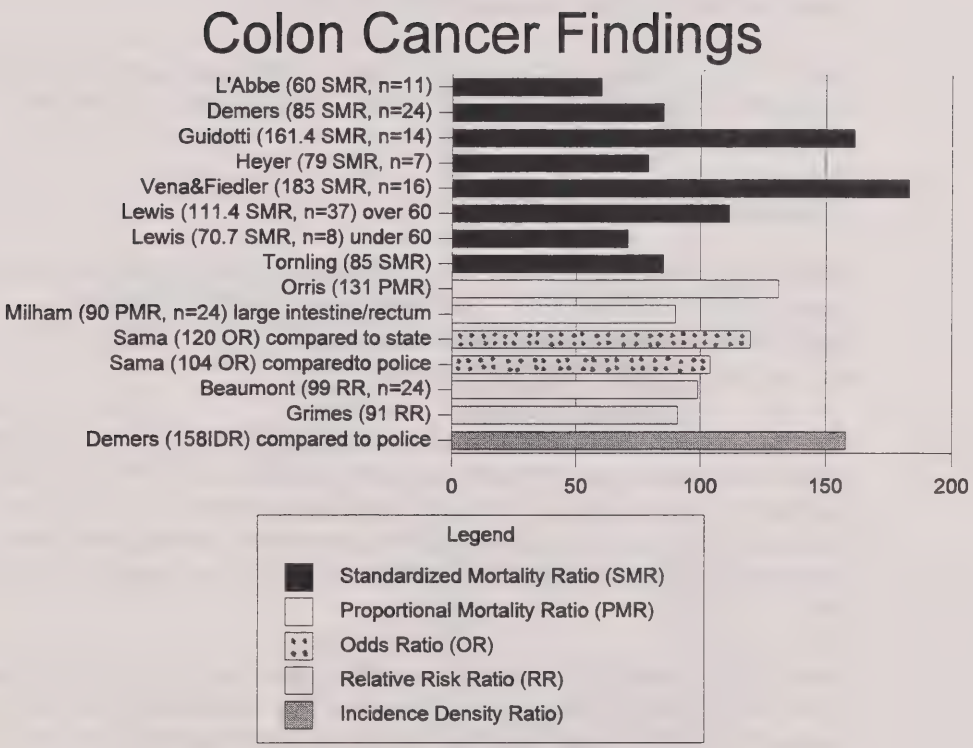
AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COM-PARISON CONTROL GROUP	GEO-GRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	LYMPHATIC AND HAEMATO-POIETIC CANCERS - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
ORRIS, et al.; (1992); proportional mortality ratio ("PMR") study	3084 white male FF deaths	1940-88 (49 yrs.)	U.S.A. white males	Chicago, Illinois, U.S.A.	Not applicable	Not applicable	not specified	Analysis done for IDSP: Leukaemia PMR for 1978-82: 2.382 ($p<0.005$); (1.191-4.224); n=11; no dose-response analysis
MUSK et al.; (1978); standardized mortality ratio ("SMR") study	5655 males	1915-75 (61 yrs.)	male population of Mass. and U.S.A.	Boston, Mass., U.S.A.	yes	2470 deaths; 91% of expected for Mass. males; 94% of expected for U.S.A. white males	SMR 63; (39-95); n=22; no dose-response analysis	
L'ABBÉ & TOMLINSON (1992); (IDSP study); standardized mortality ratio ("SMR") study	5,414 males; 5,373 for duration of employment analysis	1950-89 (40 yrs.)	Ontario males	Metropolitan Toronto	strong for FFs under 40 and for FFs with less than 10 years since first exposure	SMR 94; (88-101); n=777	SMR 98; (58-155); n=18 Lymphatic leukaemia: SMR 190; (52-485); n=4; possible dose-response Lymphosar-coma: SMR 203; (42-592); n=3	no dose-response analysis for causes other than lymphatic leukaemia

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COM-PARISON CONTROL GROUP	GEO-GRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	LYMPHATIC AND HAEMATO-POIETIC CANCERS - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
DEMERS et al.; (1992); standardized mortality ratio ("SMR") study	4546 males	1945-89 (45 yrs.)	1) U.S. white males; 2) 3676 police from Seattle & Tacoma, Wash., U.S.A. (white males)	Seattle & Tacoma, Wash, U.S.A.	yes, except for diseases of the arteries, veins & pulmonary circulation in FFs with 30 or more years of exposure	SMR 0.81; (0.77-0.86); n=1169	SMR compared to US males: 1.31; (0.92-1.81); n=37; in FFs with 30 or more years employment SMR 2.05; (1.1-3.6); n=12; Incidence Density Ratio compared to police: 1.03; (0.62-1.73); possible dose-response Leukaemia: SMR compared to US males: 1.27; (0.71-2.09); n=15; in FFs with 30 or more years of employment: SMR 2.60; (1.0-5.4); n=7; Incidence Density Ratio compared to police: 0.80; (0.38-1.70); no dose-response	compares FFs with police, who are also screened for physical health; lymphatic & haemato-poietic cancers overall, and leukaemia, were elevated among police compared to US white males
GUIDOTTI; (1993); standardized mortality ratio ("SMR") study	3328	1927-87 (61 yrs.)	Alberta males	Edmonton & Calgary, Alberta	no, probably because: 1-Alberta males are unusually healthy; and, 2-most subjects were traced	SMR 96.2; (86.6-106.5); n=370	SMR 126.5; (60.6-232.5); n=10; no dose-response analysis for these causes	
BEAUMONT et al.; (1991); mortality rate ratio ("RR") study	3066 white males	1940-82 (43 yrs.)	U.S.A. white males	San Francisco, Calif., U.S.A.	yes	RR 0.90; (0.85-0.95); n=1186	RR 0.65; (0.35-1.09); n=14; no dose-response analysis for these causes	
HEYER et al.; (1990); standardized mortality ratio ("SMR") study	2289 males	1945-83 (39 yrs.)	U.S.A. white males	Seattle, Wash., U.S.A.	yes; survivor effect also noted.	SMR 76; (69-85); n=383	SMR 126; (65-222); n=12; Leukaemia & Aleukemia: SMR 173; (70-358); n=7; other L/H: SMR 225; (47-660); n=3; -dose-response not apparent for leukaemia, but is shown for "other lymphatic/hema-topoietic" cancers	statistical-ly significant increases in FFs with 30+ years of exposure: leukaemia SMR 503; (104-1,470); n=3; "other L/H" SMR 989; (120-3,571); n=2

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COM-PARISON CONTROL GROUP	GEO-GRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	LYMPHATIC AND HAEMATO-POIETIC CANCERS - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
VENA & FIEDLER; (1987); standardized mortality ratio ("SMR") study	1867 white males	1950-79 (30 yrs.)	U.S.A. white males	Buffalo, N.Y., U.S.A.	yes, especially for circulatory system diseases.	SMR 0.95; (0.87-1.04); n=470	SMR 0.55; (0.18-1.29); n=5; no dose-response analysis for these causes	
LEWIS et al.; (1982); standardized cancer mortality ratio ("SMR") study	1559	1940-80 (41 yrs.)	U.S.A. white males	Los Angeles, Calif.	yes	declining	Leukaemia: SMR for under age 60: 63.9; (17-164); n=4; 60 and over: 91.8; (46-164); n=11; no dose-response analysis	mention of any cancer on death certificate was coded as a cancer death; this may over-estimate cancer rate
TORNLING et al.; (1994); standardized mortality ratio ("SMR") study	1116	1951-86 (36 yrs.)	Stockholm males	Stockholm, Sweden	yes	SMR 82; (73-91); n=316	SMR 44; (9-127); n=3; dose-response is shown	
DECOUFLE et al., (1977); proportionate mortality ("PMR") study	1113 stationary engineers and firefighters	1967 (1 year)	U.S. PMRs	4 areas of U.S.	not applicable	not applicable	Leukaemia: PMR 0.73; (0.27-1.59); n=6; Lymphomas: PMR 1.21; (0.64-2.07); n=13; no dose-response analysis	combines findings for engineers and firefighters which may dilute or over-estimate results
MASTRO-MATTEO; (1959); standardized mortality ratio ("SMR") study	1039	1921-53 (33 yrs.)	Toronto & Ontario males	Toronto, Ontario	not mentioned	significant excess of deaths for all causes compared to Toronto males	no significant increase or decrease in cancer deaths; no dose-response analysis	
ELIOPULOS et al.; (1984); standardized mortality ratio ("SMR") and proportionate mortality ratio ("PMR") study	990	1939-78 (about 40 yrs.)	Western Australian males	Western Australia	yes	SMR 0.80; (0.67-0.96); n=116	PMR 1.88; (0.39-5.50); n=3; no dose-response analysis	

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COM-PARISON CONTROL GROUP	GEO-GRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	LYMPHATIC AND HAEMATO-POIETIC CANCERS - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
SAMA et al.; (1990); case-control study	321 FF cancer cases	1982-86 (5 yrs.)	1) 29,277 Massachusetts males; 2) 392 police	state of Massachusetts, U.S.A.	not applicable	not applicable	Leukaemia: Odds Ratio compared to police: 267; (62-1154); compared to state: 112; (48-259); n=6 Lymphoma: Odds Ratio compared to police: 327; (119-898); compared to state: 159; (89-284); n=14; Odds Ratio for ages 55-74: 538 (150-1924); n=10; no dose-response analysis	compares FFs with state population and with police, who are also screened for physical health
FEUER & ROSENMAN; (1986); proportional mortality ratio ("PMR") study	263 FF deaths	1974-80 (7 yrs.)	1) U.S. PMRs; 2) N.J. PMRs; 3) N.J. police PMRs	state of New Jersey, U.S.A.	not applicable	not applicable	Leukaemia: PMR compared to U.S. PMR: 1.86; (0.51-4.76); compared to N.J. PMR: 1.77; (0.48-4.53); compared to N.J. police PMR: 2.76; (0.75-7.07); ($p<0.05$); n=4; no dose-response analysis for this cause	compares FFs with general U.S. and state popula-tions and with police, who are also screened for physical health; rate of leukaemia among police was decreased
GRIMES et al.; (1991); proportional mortality ratio ("PMR") study	205 males	1969-88 (20 yrs.)	20 year and older Hawaiian males	Honolulu, Hawaii, U.S.A.	mentioned by authors	not applicable	Lymphatic system: Risk Ratio: 0.95; (0.36-2.50); no dose-response analysis	
MILHAM; (1983); proportional mortality ratio ("PMR") study	not stated	1950-79 (30 yrs.)	Washington state males	state of Washington, U.S.A.	not applicable	not applicable	PMR 135; (96-185); n=39; no dose-response analysis	

Fig. 18


Colon Cancer Findings
L'Abbe (60 SMR, n=11)
Demers (85 SMR, n=24)
Guidotti (161.4 SMR, n=14)
Heyer (79 SMR, n=7)
Vena&Fiedler (183 SMR, n=16)
Lewis (111.4 SMR, n=37) over 60
Lewis (70.7 SMR, n=8) under 60
Tornling (85 SMR)
Orris (131 PMR)
Milham (90 PMR, n=24) large intestine/rectum
Sama (120 OR) compared to state
Sama (104 OR) comparedto police
Beaumont (99 RR, n=24)
Grimes (91 RR)
Demers (158IDR) compared to police
0
50
100
150
200
Legend
Standardized Mortality Ratio (SMR)
Proportional Mortality Ratio (PMR)
Odds Ratio (OR)
Relative Risk Ratio (RR)
Incidence Density Ratio)

(v) Colon cancer

It is estimated that in 1993 colorectal cancer accounted for 14% of new cancer cases and 10.1% of cancer deaths among adult males in Canada [130].

Mortality and morbidity studies

Three studies identified statistically significant increases in colon cancer mortality among firefighters [147, 202, 13]. Five studies provided dose-response analyses for cancer of the colon (or "intestine") [59, 10, 32, 202, 86]; two of them suggested a dose-response trend and showed increases with longer latency [32, 202]. Four other studies found increases that were not statistically significant [59, 169, 32, 98], and seven found no increase or fewer deaths than expected from this cause [10, 121, 56, 68, 86, 195, 109]. Four of the studies did not report findings for colon cancer [128, 43, 63, 47].

The findings of thirteen studies of colon cancer in firefighters are illustrated in Figure 18. More detail can be found in Table 14 which appears at the end of this section.

Statistically significant increases

Orris and co-workers (1992) reported on the causes of 3084 Chicago firefighter deaths between 1940 and 1988. That group experienced a statistically significant increase in cancer of the intestine (PMR 131; 95% CI 104-165), a broader category which includes the colon.

Vena and Fiedler (1987) identified a statistically significant increase in colon cancer mortality among 1867 firefighters from Buffalo, New York. The SMR reached 183 (95% CI 105-297; $p<0.05$) based upon sixteen deaths. The rate increased with longer latency and a dose-response trend was suggested, with a statistically significant SMR of 471 in those with the longest employment (Fig. 18).

Since it is not a study of firefighters specifically, Berg and Howell's 1975 study of bowel cancer is not described in Table 14. These authors analyzed US data gathered in 1950 on occupational mortality among males aged 20-64 and identified an SMR of 279 ($p<0.01$) for cancer of the "bowel" among firefighters. Although ICD codes are not given, "bowel" usually refers to the large intestine. The authors' analysis led them to conclude that "while an environmental factor may preferentially affect one segment of the large bowel, it acts to some extent on both rectum and colon. Occupational hazards, then, would not be expected to differ markedly for the two sites". This study thus appears to support an association, prior to 1950, between firefighting and colon cancer.

FIG. 19: COLON CANCER MORTALITY AMONG BUFFALO FIREFIGHTERS, 1950-79, BY DURATION OF EMPLOYMENT:

Years worked	SMR	(95%CI)	n
1-9	0.00		0
10-19	1.25	(0.0-4.901	1
20-29	0.87	(0.082-2.492)	2
30-39	1.43	(0.451-2.955)	5
40+	4.71	(2.010-8.532)*	8
Total	1.83	(1.048-2.851)*	16

*p<0.05
n=number of deaths
(Vena & Fiedler, 1987)

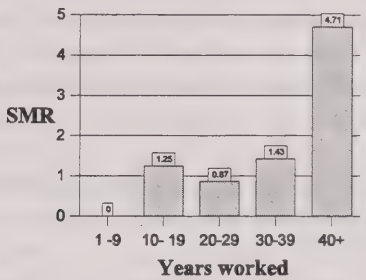
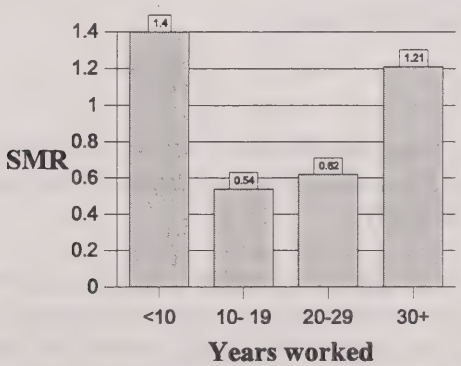


FIG. 20: COLON CANCER MORTALITY AMONG FIREFIGHTERS FROM THREE NORTHWESTERN U.S. CITIES, 1945-89, BY DURATION OF EMPLOYMENT:

Years worked	SMR	(95%CI)	n
<10	1.40	(0.4-3.6)	4
10-19	0.54	(0.1-2.0)	2
20-29	0.62	(0.3-1.2)	9
30+	1.21	(0.6-2.3)	9

n=number of deaths
(Demers et al, 1992)



Other findings

Of the studies which found increases which were not statistically significant, the largest involved 3328 Alberta firefighters studied by Guidotti (1993). He reported an SMR from colon and rectum cancer combined of 161.4 based upon fourteen deaths (95% CI 88.3-270.9).

A case-control study of cancer incidence by Sama et al. (1990) involved 321 firefighters from Massachusetts. They found an Odds Ratio of 120 (95% CI 80-182) compared with males from that state, but the OR dropped to 104 (95% CI 59-182) when police officers were used as the comparison group.

In contrast, the Demers (1992) firefighters from the northwestern US had a lower rate of colon cancer than US males but a considerably higher rate than police. The firefighters' higher rate is perhaps partly attributable to the statistically significant decrease in the rate of colon cancer among the police.

Seven studies found no increase or fewer deaths than expected from this cause [10, 121, 56, 68, 195,109], including the IDSP study which found an SMR of 60 (95% CI 30-108).

Dose-response analyses

As mentioned above, Vena and Fiedler noted a dose-response trend among Buffalo firefighters (Fig. 19). Such a trend was also possible in the study by Demers et al. (1992) among northwestern US firefighters (Fig. 20). Three other studies conducted dose-response analyses but no such trend was found [59, 10, 86].

Potential causative agents

Asbestos

IARC has classified asbestos as Group 1 for which there is sufficient evidence to establish its carcinogenicity to humans in the case of colon cancer, among other cancers [79].

Because of its insulating properties, asbestos had been widely used in building materials for residential, commercial and industrial settings. As noted under "Chemical Hazards of Firefighting" above, Markowitz et al. (1980) reported strong evidence that firefighters are at risk for scarring of the lungs and pleura due to occupational asbestos exposure [107].

Diesel exhaust

Diesel exhaust is described as a probable carcinogen (Group 2A) to humans by IARC [80]. Although there is no direct evidence to link diesel exhaust exposure to colon cancer in firefighters, exposure to PAHs, which are present in diesel exhaust, has been linked with colon cancer among coke oven workers [100]. Firefighters may be at increased risk because of frequent and repeated exposure to diesel exhaust [97, 49, 28].

Other

IARC describes acrylonitrile and formaldehyde as probable carcinogens (Group 2A) associated with colon cancer and formaldehyde [79].

Both acrylonitrile and formaldehyde may be present in the air at fire sites as combustion products of building and insulation materials. None of the studies reviewed conducted chemical analyses for acrylonitrile in fire smoke; however, the Panel's Industrial Hygienist advises that firefighters could be frequently exposed to acrylonitrile vapours generated from thermal decomposition of various rubber products. As for formaldehyde, significant concentrations have been measured in studies of different types of fires [19, 82].

Summary of the evidence

- Statistically significant increases in colon cancer were identified in three studies. One of those increases occurred among the largest cohort, but the finding was for the broader category of cancer of the intestine.

- Increases did not appear consistently which weakens the strength of the association between firefighting and colon cancer.

- Firefighters experience occupational exposure to carcinogens which can cause colon cancer such as asbestos, PAHs, acrylonitrile and formaldehyde.

- Dose-response trends were shown in a minority of the studies which conducted that analysis. Since the actual occupational exposures of the firefighters studied is unknown, negative dose-response findings are not evidence against an association, but are simply uninformative.

The Panel's conclusions and finding

While there is evidence for and against, the Panel concludes that the weight of the evidence establishes a probable connection between firefighting and colon cancer. The association is strong in three studies, one of which is of the largest cohort, but there is a lack of consistency in the findings. There is some evidence of dose-response trends and evidence that firefighters are exposed to recognized colon carcinogens.

Accordingly, the Panel makes the following finding.

- **A probable connection exists between colon cancer and the occupation of firefighting.**

Since there is a lack of consistency in the data, the evidence did not persuade the Panel to recommend inclusion in a Schedule to the Regulations of the *Act* which would invoke a legal presumption that colon cancers in firefighters are due to their occupation, unless the contrary is proved.

The Panel's recommendation

- **Because a probable connection has been established, guidelines developed and approved by the Panel should be used to assist adjudicators in assessing the merits of each claim for colon cancer from firefighters.**

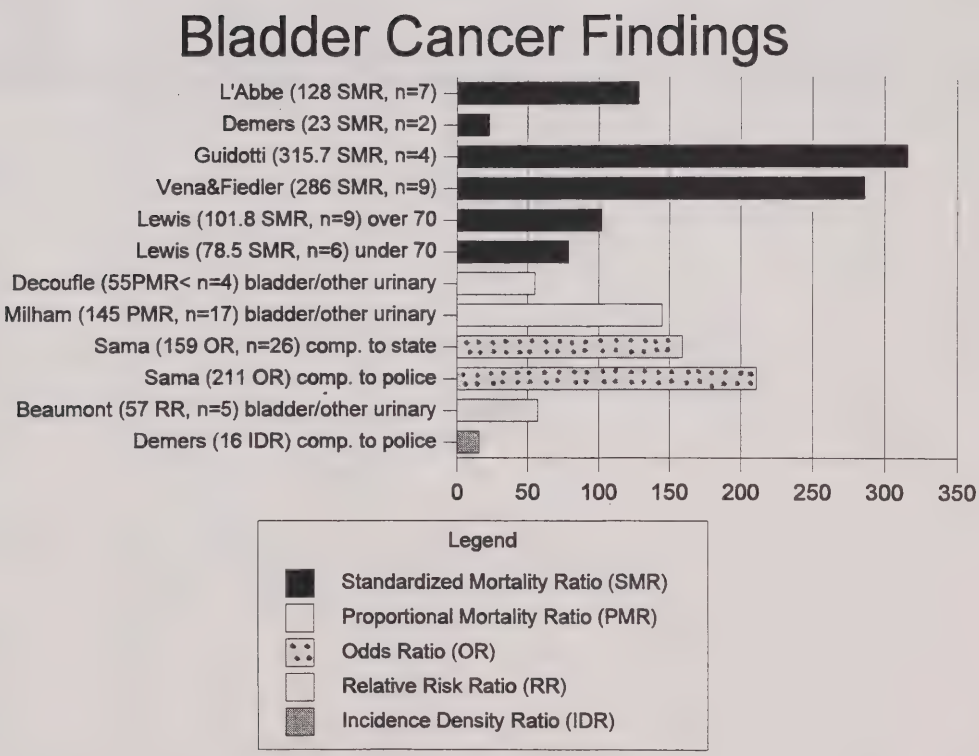
TABLE 14: FIREFIGHTER MORTALITY/MORBIDITY STUDIES: COLON CANCER FINDINGS

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	COLON CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
ORRIS, et al.; (1992); proportional mortality ratio ("PMR") study	3084 white male FF deaths	1940-88 (49 yrs.)	U.S.A. white males	Chicago, Illinois, U.S.A.	Not applicable	Not applicable	Intestine: PMR 131; (104-165); n=not given; no dose-response analysis	
L'ABBÉ & TOMLIN-SON; (1992); (IDSP study); standardized mortality ratio ("SMR") study	5,414 males; 5,373 for duration of employment analysis	1950-89 (40 yrs.)	Ontario males	Metropolitan Toronto	strong for FFs under 40 and for FFs with less than 10 years since first exposure	SMR 94; (88-101); n=777	Colon: SMR 60; (30-108); n=11; dose-response not apparent	
DEMERS et al.; (1992); standardized mortality ratio ("SMR") study	4546 males	1945-89 (45 yrs.)	1) U.S. white males; 2) 3676 police from Seattle & Tacoma, Wash., U.S.A. (white males)	Seattle & Tacoma, Wash, U.S.A.	yes, except for diseases of the arteries, veins & pulmonary circulation in FFs with 30 or more years of exposure	SMR 0.81; (0.77-0.86); n=1169	Colon: SMR 0.85; (0.54-1.26); n=24; Incidence Density Ratio compared to police: 1.58; (0.73-3.43); possible dose-response	com-pares FFs with police, who are also screen-ed for physical health
GUIDOTTI; (1993); standardized mortality ratio ("SMR") study	3328	1927-87 (61 yrs.)	Alberta males	Edmonton & Calgary, Alberta	no, probably because: 1-Alberta males are unusually healthy; and, 2-most subjects were traced	SMR 96.2; (86.6-106.5); n=370	Colon and rectum: SMR 161.4; (88.3-270.9); n=14; dose-response not apparent	
BEAUMONT et al.; (1991); mortality rate ratio ("RR") study	3066 white males	1940-82 (43 yrs.)	U.S.A. white males	San Francisco, Calif., U.S.A.	yes	RR 0.90; (0.85-0.95); n=1186	Intestine except rectum: RR 0.99; (0.63-1.47); n=24; dose-response not shown	

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	COLON CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COMMENTS
HEYER et al.; (1990); standardized mortality ratio ("SMR") study	2289 males	1945-83 (39 yrs.)	U.S.A. white males	Seattle, Wash., U.S.A.	yes; survivor effect also noted.	SMR 76; (69-85); n=383	Intestine: SMR 79; (32-164); n=7; no dose-response analysis for this cause	
VENA & FIEDLER; (1987); standardized mortality ratio ("SMR") study	1867 white males	1950-79 (30 yrs.)	U.S.A. white males	Buffalo, N.Y., U.S.A.	yes, especially for circulatory system diseases.	SMR 0.95; (0.87-1.04); n=470	Colon: SMR 1.83; (1.05-2.97); ($p<0.05$); n=16; possible dose-response	
LEWIS et al.; (1982); standardized cancer mortality ratio ("SMR") study	1559	1940-80 (41 yrs.)	U.S.A. white males	Los Angeles, Calif.	yes	declining	Colon: SMR for under age 60: 70.7; 31-139); n=8; 60 and over: 111.4; (78-154); n=37; no dose-response analysis	mention of any cancer on death certificate was coded as a cancer death; this may overestimate cancer rate
TORNLING et al.; (1994); standardized mortality ratio ("SMR") study	1116	1951-86 (36 yrs.)	Stockholm males	Stockholm, Sweden	yes	SMR 82; (73-91); n=316	Small intestine and colon: SMR 85; (31-185); n=6; no dose-response analysis for this cause	
MASTRO-MATTEO; (1959); standardized mortality ratio ("SMR") study	1039	1921-53 (33 yrs.)	Toronto & Ontario males	Toronto, Ontario	not mentioned	significant excess of deaths for all causes compared to Toronto males	no significant increase or decrease in cancer deaths; no dose-response analysis	
SAMA et al.; (1990); case-control study	321 FF cancer cases	1982-86 (5 yrs.)	1) 29,277 Massachusetts males; 2) 392 police	state of Massachusetts, U.S.A.	not applicable	not applicable	Colon: Odds Ratio compared to police: 104; (59-182); compared to state: 120; (80-182); n=33; no dose-response analysis	compares FFs with general population and with police, who are also screened for physical health

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	COLON CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
GRIMES et al.; (1991); propo-tional mortality ratio ("PMR") study	205 males	1969-88 (20 yrs.)	20 year and older Hawaiian males	Honolulu, Hawaii, U.S.A.	mentioned by authors	not applicable	Colon: Risk Ratio 0.91; (0.37-2.20); no dose-response analysis	
MILHAM; (1983); proportional mortality ratio ("PMR") study	not stated	1950-79 (30 yrs.)	Washington state males	state of Washington, U.S.A.	not applicable	not applicable	Large intestine except rectum: PMR 90; (58-134); n=24; no dose-response analysis	

Fig. 21


Bladder Cancer Findings
L'Abbe (128 SMR, n=7)
Demers (23 SMR, n=2)
Guidotti (315.7 SMR, n=4)
Vena&Fiedler (286 SMR, n=9)
Lewis (101.8 SMR, n=9) over 70
Lewis (78.5 SMR, n=6) under 70
Decoufle (55PMR< n=4) bladder/other urinary
Milham (145 PMR, n=17) bladder/other urinary
Sama (159 OR, n=26) comp. to state
Sama (211 OR) comp. to police
Beaumont (57 RR, n=5) bladder/other urinary
Demers (16 IDR) comp. to police
0
50
100
150
200
250
300
350
Legend
Standardized Mortality Ratio (SMR)
Proportional Mortality Ratio (PMR)
Odds Ratio (OR)
Relative Risk Ratio (RR)
Incidence Density Ratio (IDR)

(vi) Bladder cancer

It is estimated that cancer of the bladder accounted for 6.1% of new cancer cases and 2.8% of cancer deaths among adult males in Canada in 1993 [130].

Mortality and morbidity studies

Findings from thirteen studies for bladder cancer in firefighters are illustrated in Figure 21. More detail appears in Table 15, at the end of this section.

Three studies, each using a different method, found statistically significant increases in bladder cancer among firefighters, reported by Vena and Fiedler (1987), Milham (1983) and Sama and colleagues (1990). One study indicated a dose-response trend, reported by Vena and Fiedler (1987). Five found increases which did not reach statistical significance [86, 59, 10, 98, 121].

Three authors did not provide findings for bladder cancer alone, but only reported findings for the broader category of "genitourinary" cancer, which includes cancers of the prostate, testis, kidney, bladder, etc. Since cancer of the prostate accounts for about half of the cancers in this broad category, those three studies are not included in the discussion below [128, 43, 56].

One large mortality study found a statistically significant decrease compared to the general population [32]. Another study reported a decrease which was not statistically significant [31] but it combined firefighters with stationary engineers which renders those findings less informative.

Statistically significant increases

Sama and co-workers (1990) conducted a case-control study involving 321 cases of cancer among Massachusetts firefighters using that state's Cancer Registry files for 1982 through 1986. They found a statistically significant Odds Ratio of 159 for bladder cancer among firefighters compared to Massachusetts males (95% CI 102-250) and found an even higher rate when firefighters were compared to police officers (OR 211; 95% CI 107-414; n=26). Employment status was determined from the usual occupation reported to the Registry, however, since occupation was available for only about 50% of all Registry cases, the authors thought that the findings could underestimate the true incidence of firefighters' cancer by as much as half. Smoking is known to be associated with bladder cancer but since the lung cancer rate was not also elevated, the authors concluded that the excess of bladder cancer was unlikely to be entirely smoking-related.

FIG. 22: BLADDER CANCER MORTALITY AMONG BUFFALO FIREFIGHTERS, 1950-1979, BY DURATION OF EMPLOYMENT:

Years worked	SMR	(95%CI)	n
1-9	5.00	(0.00-19.60)	1
10-19	0.00		0
20-29	1.25	(0.00-4.90)	1
30-39	2.14	(0.40-5.25)	3
40+	5.71	(1.49-12.69)*	4
Total	2.84*	(1.27-4.95)	9

*p=<0.05
n=number of deaths
(Vena and Fiedler, 1987)

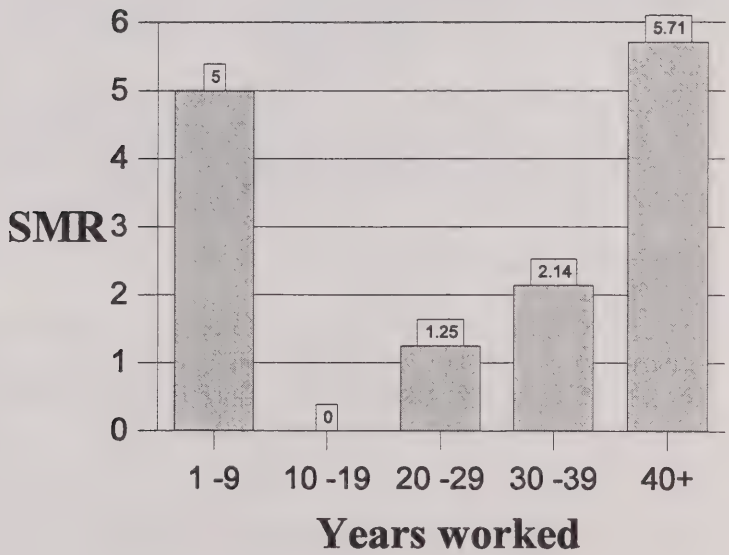


FIG.

Vena and Fiedler (1987) reported a statistically significant increase in the SMR for bladder cancer among 1867 firefighters from Buffalo, New York (SMR 286; 95% CI 130-540; n=9).

A statistically significant increase in the proportional mortality ratio for bladder cancer was also found by Milham (1983) among firefighters from Washington state (PMR 233; $p<0.05$; n=17).

Other increases

Guidotti (1993) found an increase in bladder cancer among 3328 Alberta firefighters, but the overall rate for the whole cohort did not reach statistical significance based upon four cases (SMR 315.7; 95% CI 86-808.3).

The IDSP study of 5414 Toronto firefighters found seven bladder cancer deaths with an SMR of 128 (95% CI 51-263) but no dose-response trend appeared.

Other findings

Three other studies found no increase or found a decrease [10, 32, 195]. In the report by Demers et al. (1992), the decrease was statistically significant compared to the general population (SMR 23; 95% CI 3-83). The decrease persisted when police were used as the reference group but was not statistically significant (IDR 16; 95% CI 02-124).

Dose-Response Analyses

As noted above, one study [202] suggested a dose-response trend (Fig. 22) but two others did not [86, 59]. Although no dose-response trend was shown in the Alberta cohort studied by Guidotti (1993), the peak risk, observed 40 to 49 years after entry into the fire service, was statistically significant (SMR 1392.8; 95% CI 287.4-4070.3; $p<0.01$). The other studies did not conduct dose-response analyses for bladder cancer.

Potential causative agents

Diesel Exhaust

IARC reports that diesel exhaust is a probable carcinogen (Group 2A) associated with bladder cancer in humans [80].

Several studies have shown an increased risk of bladder cancer in truck drivers and attributed the cause to diesel exhaust exposure [177, 70, 221]. Firefighters

may also be at increased risk because of their potentially frequent and repeated exposure to diesel exhaust [97, 49, 28].

Formaldehyde

IARC describes formaldehyde as a probable carcinogen (Group 2A) associated with bladder cancer [79].

Formaldehyde is used in the manufacture of various household products and building materials [20, 164]. It is therefore likely to be present in fires as a thermal decomposition product of such materials. Concentrations high enough to cause adverse acute health effects have been detected in both content and building fires [19, 82].

Summary of the evidence

- Statistically significant increases in bladder cancer were identified in some firefighter cohorts, with very high relative risks.

- Since the relative five-year survival rate for bladder cancer is estimated to be 71% [130], studies based on death certificate data will not be as reliable as morbidity studies. Consequently, the Panel gives particular weight to the 1990 cancer incidence study by Sama and colleagues. The study design took steps to overcome the problems arising from reliance on death certificate data and attempted to control for the healthy worker effect by comparing firefighters with another occupational group that is screened for physical health. It found a significant increase among firefighters compared to police officers as well as state males.

- Statistically significant increases did not appear consistently among the mortality data and one statistically significant decrease was reported. This lack of consistency weakens the strength of the association between firefighting and bladder cancer.

- Only one of the three studies which conducted the analysis suggested a dose-response trend. As stated above, however, the use of duration of employment as a surrogate for exposure does not provide meaningful information about "dose", hence the lack of a dose-response trend is not surprising.

- Firefighters experience occupational exposure to bladder carcinogens such as formaldehyde, diesel exhaust and PAHs.

The Panel's conclusions and finding

These data led the Panel to conclude that there is evidence, although not consistent evidence, of a probable connection between firefighting and bladder cancer. Since the survival rate for bladder cancer is high, the cancer incidence study provides particularly strong and reliable evidence. There is some evidence of a dose-response. A probable connection is biologically plausible since firefighters are exposed to bladder carcinogens.

Accordingly, the Panel makes the following finding.

- **A probable connection exists between primary bladder cancer and the occupation of firefighting.**

Since there is a lack of consistency in the data, the evidence did not persuade the Panel to recommend inclusion in a Schedule to the *Act* which would invoke a legal presumption that bladder cancers in firefighters are due to their occupation, unless the contrary is proved.

The Panel's recommendation

- **Because a probable connection has been established, guidelines developed and approved by the Panel should be used to assist adjudicators in assessing the merits of claims for bladder cancer from firefighters.**

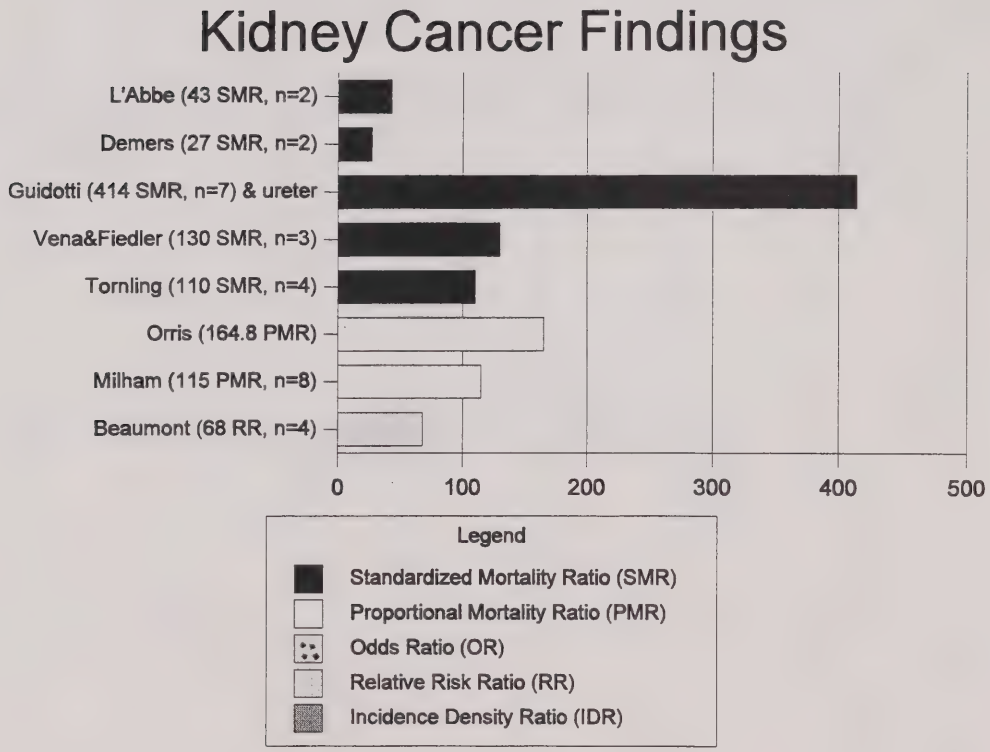
TABLE 15: FIREFIGHTER MORTALITY/MORBIDITY STUDIES: BLADDER CANCER FINDINGS

AUTHORS; YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	BLADDER CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COMMENTS
L'ABBÉ & TOMLIN-SON; (1992); (IDSP study); standardized mortality ratio ("SMR") study	5,414 males; 5,373 for duration of employment analysis	1950-89 (40 yrs.)	Ontario males	Metropolitan Toronto	strong for FFs under 40 and for FFs with less than 10 years since first exposure	SMR 94; (88-101); n=777	Bladder: SMR 128; (51-263); n=7; no dose-response	
DEMERS et al.; (1992); standardized mortality ratio ("SMR") study	4546 males	1945-89 (45 yrs.)	1) U.S. white males; 2) 3676 police from Seattle & Tacoma, Wash., U.S.A. (white males)	Seattle & Tacoma, Wash, U.S.A.	yes, except for diseases of the arteries, veins & pulmonary circulation in FFs with 30 or more years of exposure	SMR 0.81; (0.77-0.86); n=1169	Bladder and other urinary: SMR compared to US males: 0.23; (0.03-0.83); n=2; Incidence Density Ratio compared to police: 0.16; (0.02-1.24); no dose-response analysis for this cause	com-pares FFs with police, who are also scr-eened for physical health
GUIDOTTI; (1993); standardized mortality ratio ("SMR") study	3328	1927-87 (61 yrs.)	Alberta males	Edmonton & Calgary, Alberta	no, probably because: 1-Alberta males are unusually healthy; and, 2-most subjects were traced	SMR 96.2; (86.6-106.5); n=370	Bladder: SMR 315.7; (91.0-211.4); n=4; dose-response not apparent	
BEAUMONT et al.; (1991); mortality rate ratio ("RR") study	3066 white males	1940-82 (43 yrs.)	U.S.A. white males	San Francisco, Calif., U.S.A.	yes	RR 0.90; (0.85-0.95); n=1186	Bladder and other urinary: Rate Ratio 0.57; (0.19-1.35); n=5; no dose-response analysis for this cause	
VENA & FIEDLER; (1987); standardized mortality ratio ("SMR") study	1867 white males	1950-79 (30 yrs.)	U.S.A. white males	Buffalo, N.Y., U.S.A.	yes, especially for circulatory system diseases.	SMR 0.95; (0.87-1.04); n=470	Bladder: SMR 2.86; (1.30-5.40) $p<0.05$; n=9; possible dose-response	
LEWIS et al.; (1982); standardized cancer mortality ratio ("SMR") study	1559	1940-80 (41 yrs.)	U.S.A. white males	Los Angeles, Calif.	yes	declining	Bladder: SMR for under age 70: 78.5; (29-171); n=6; 70 and over: 101.8; (47-193); n=9; no dose-response analysis	mention of any cancer on death certificate was coded as a cancer death; this may overestim ate cancer rate

AUTHORS; YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	BLADDER CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COMMENTS
LEWIS et al.; (1982); standardized cancer mortality ratio ("SMR") study	1559	1940-80 (41 yrs.)	U.S.A. white males	Los Angeles, Calif.	yes	declining	Bladder: SMR for under age 70: 78.5; (29-171); n=6; 70 and over: 101.8; (47-193); n=9; no dose-response analysis	mention of any cancer on death certificate was coded as a cancer death; this may overestimate cancer rate
DECOUFLE et al., (1977); proportionate mortality ("PMR") study	1113 stationary engineers and firefight-ers	1967 (1 year)	U.S. PMRs	4 areas of U.S.	not applicable	not applicable	Bladder and other urinary: PMR 0.55; (15-141); n=4; no dose-response analysis	no confidence intervals given; com-bines findings for engineers and firefighters which may dilute or overestimate results
MASTROMATTEO; (1959); standardized mortality ratio ("SMR") study	1039	1921-53 (33 yrs.)	Toronto & Ontario males	Toronto, Ontario	not mentioned	significant excess of deaths for all causes compared to Toronto males	no significant increase or decrease in cancer deaths; no dose-response analysis	
SAMA et al.; (1990); case control study	321 FF cancer cases	1982-86 (5 yrs.)	1) 29,277 Massachusetts males; 2) 392 police	state of Massachusetts, U.S.A.	not applicable	not applicable	Bladder: OR compared to police: 211; (107-414); compared to state: 159; (102-250); n=26; no dose-response analysis	compares FFs with general population and with police, who are also screened for physical health
MILHAM; (1983); proportional mortality ratio ("PMR") study	not stated	1950-79 (30 yrs.)	Washington state males	state of Washington, U.S.A.	not applicable	not applicable	Bladder and other urinary: PMR 145; (84-232); n=17; no dose-response analysis	statistically significant increase in ages 20-64: PMR 233 ($p<0.05$); n=6

AUTHORS; YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	BLADDER CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COMMENTS
GRIMES et al.; (1991); proportional mortality ratio ("PMR") study	205 males	1969-88 (20 yrs.)	20 year and older Hawaiian males	Honolulu, Hawaii, U.S.A.	yes, mentioned by authors	not applicable	"Genito-urinary system": Risk Ratio 2.28; (1.28-4.06); no dose-response analysis	"genito-urinary" includes bladder, kidney, ureter, prostate, testis and others

Fig. 23


Kidney Cancer Findings
L'Abbe (43 SMR, n=2)
Demers (27 SMR, n=2)
Guidotti (414 SMR, n=7) & ureter
Vena&Fiedler (130 SMR, n=3)
Tornling (110 SMR, n=4)
Orris (164.8 PMR)
Milham (115 PMR, n=8)
Beaumont (68 RR, n=4)
0
100
200
300
400
500
Legend
Standardized Mortality Ratio (SMR)
Proportional Mortality Ratio (PMR)
Odds Ratio (OR)
Relative Risk Ratio (RR)
Incidence Density Ratio (IDR)

(vii) Kidney cancer

It is estimated that cancer of the kidney accounted for 3.4% of new cancer cases and 2.5% of cancer deaths among adult males in Canada in 1993 [130].

Mortality and morbidity studies

The findings of twelve studies of kidney and genito-urinary cancer in firefighters are illustrated in Figure 23. More detail is given in Table 16 which appears at the end of this section.

Two authors found statistically significant increases in kidney cancer mortality among firefighters [59, 190] and three found elevations which were not statistically significant [202, 195, 121]. Two studies showed possible dose-response trends [59, 195]; in a third, the Panel concluded that no dose-response trend occurred [86]. Four found no increase or a decrease in rates of kidney/genito-urinary cancers [86, 10, 32,109]. In one of them, the rate of kidney cancer showed a statistically significant decrease [32].

Three other authors reported findings for the broad category of "genito-urinary" mortality among firefighters [128, 43, 56]. Since "genito-urinary" includes cancer of the prostate, which would account for about half the deaths in that category, the findings of these three studies are not sufficiently specific about kidney cancer to be included in the discussion below.

Statistically significant increases

Deaths from cancers of the kidney and ureter reached statistical significance above the expected among 3328 Alberta firefighters studied by Guidotti (1993) (SMR 414; 95% CI 166.4-853.1; $p<0.05$; n=7).

Orris and colleagues (1992), who conducted the largest firefighter mortality study, did not specify results for kidney cancer. The Panel asked them to re-examine their data and provide results for several types of cancer, including kidney cancer. They found a statistically significant increase overall in kidney cancer among 3315 Chicago firefighter deaths compared to US males between 1948 and 1982 (PMR 1.648; 95% CI 1.058-2.452) [190].

Other findings

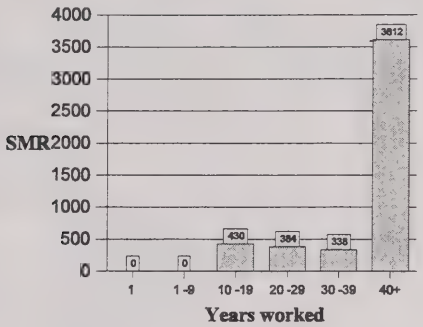
Three authors report elevations which were not statistically significant [202, 195, 121].

In contrast, Demers et al. (1992) found a statistically significant decrease in the rate of cancer of the kidney compared to the general population among 4546

Fig. 24: KIDNEY AND URETER CANCER MORTALITY AMONG ALBERTA FIREFIGHTERS, 1927-1987, BY DURATION OF EMPLOYMENT AND BY EXPOSURE OPPORTUNITY:

Years worked	SMR	n
<1	0	0
1-9	0	0
10-19	430	1
20-29	384	2
30-39	338	2
40+	3612*	2

*p<0.01
n=number of deaths



Exposure Opp.	SMR	n
0	890	1
>0,<1		0
1-4		0
5-9		0
10-14	854	1
15-19	654	1
20-24		0
25-29	522	2
30-35		0
35+	3542*	2

*p<0.01
n=number of deaths
(Guidotti, 1992)

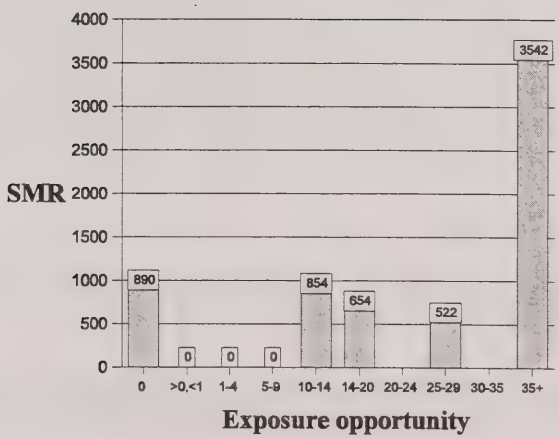
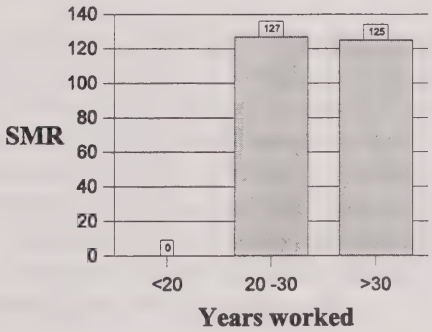


FIG. 25: KIDNEY CANCER MORTALITY AMONG SWEDISH FIREFIGHTERS, 1951-1986, BY DURATION OF EMPLOYMENT AND BY NUMBER OF FIRES ATTENDED:

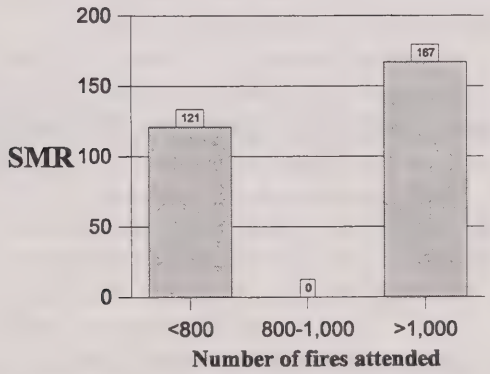
Years worked	SMR	(95% CI)	n
<20	0	(0-785)	0
20-30	127	(11-459)	2
>30	125	(15-450)	2

n=number of deaths



Years worked	SMR	(95% CI)	n
<800	121	(3-674)	1
800-1,000	0	(0-358)	0
>1,000	167	(35-489)	3

n=number of deaths
(Tornling et al., 1990)



firefighters from the northwestern US (SMR 27; 95% CI 3-97). The authors of four other studies reported no increase or decreases which were not statistically significant, including the IDSP study which found an SMR of 43 (95% CI 5-156) [86, 10, 32, 109].

As mentioned above, three authors reported findings for the broad category "genito-urinary" cancers. Grimes et al. (1991) found a statistically significant increase. Musk and colleagues. (1978) and Eliopulos et al. (1984) found about the expected. These findings include prostate cancer, among others, hence they are less than informative about cancer of the kidney among firefighters.

Dose-response analyses

Kidney and ureter cancer mortality was highest among firefighters with longest duration of employment and exposure opportunity in the Alberta firefighters studied by Guidotti (1993) (Fig. 24), although the findings did not increase consistently throughout either analysis. The author suggested that the excess of cancers of the bladder in this and other studies [202, 169], combined with the kidney and ureter cancer findings in his study are "consistent with inhaled constituents of smoke, particularly polycyclic aromatic hydrocarbons".

Although none of the rates reached statistical significance, a dose-response trend was possible in the Swedish firefighters studied by Tornling et al. (1994) (Fig. 25).

No dose-response trend appeared in the findings of the IDSP study [86].

Potential causative agents

Polycyclic aromatic hydrocarbons (PAHs)

Guidotti (1993) states that cancers of the bladder, kidney and ureter are consistent with inhaled constituents of smoke, particularly PAHs. These particulates have been measured in detectable amounts in the smoke of building fires [82]. Firefighters may also be exposed to PAHs in diesel exhaust from fire engines [97, 49, 28].

Other

IARC reports that chloroform is possibly carcinogenic to humans (Group 2B); however, the evidence of carcinogenicity to animals is described as sufficient [79].

Both chloroform and formaldehyde were measured in detectable concentrations in the smoke of various fires studied by Brandt-Rauf et al. (1988).

Summary of the evidence

- Statistically significant increases in kidney cancer were identified among two large firefighter cohorts, one of which had a very high SMR of 414.

- Statistically significant increases did not appear consistently in the mortality data and one statistically significant decrease was reported. This lack of consistency weakens the strength of the association between firefighting and kidney cancer.

- Firefighters experience occupational exposure to diesel exhaust containing PAHs, which may be linked with cancers of the bladder, kidney and ureter.

- An increase in mortality with longer duration of employment appeared in one of the three studies for which dose-response analyses are available. In another, a dose-response trend was possible. As stated previously, the use of duration of employment as a surrogate for exposure does not provide meaningful information about "dose", hence the lack of a dose-response trend in the third is also not meaningful.

The Panel's conclusions and finding

The Panel concludes that there is evidence, although not consistent evidence, of a probable connection between firefighting and kidney cancer. The statistical association in some studies is very strong. There is some evidence of a dose-response. A probable connection is biologically plausible in light of firefighters' occupational exposures. Overall, the evidence in support of a probable connection is greater than the evidence against.

Accordingly, the Panel makes the following finding.

- **A probable connection exists between kidney cancer and the occupation of firefighting.**

Since there is a lack of consistency in the data, the evidence did not persuade the Panel to recommend inclusion in a Schedule to the *Act* which would invoke a legal presumption that kidney cancers in firefighters are due to their occupation, unless the contrary is proved.

The Panel's recommendation

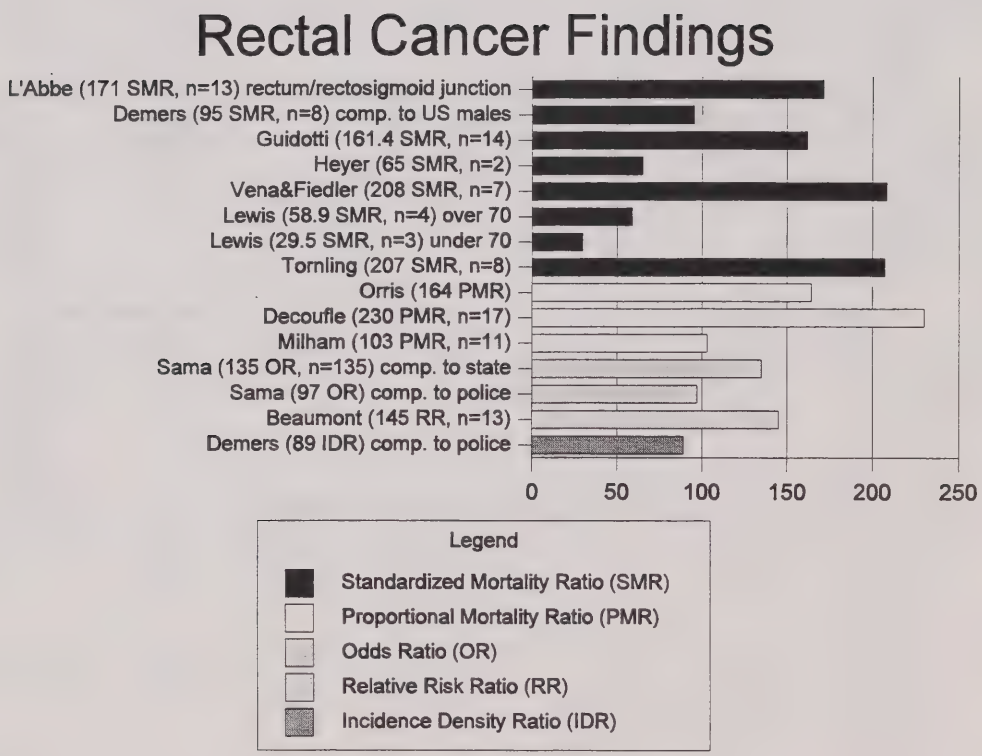
- **Because a probable connection has been established, guidelines developed and approved by the Panel should be used to assist adjudicators in assessing the merits of claims for kidney cancer from firefighters.**

TABLE 16: FIREFIGHTER MORTALITY/MORBIDITY STUDIES: KIDNEY CANCER FINDINGS

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	KIDNEY CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
ORRIS, et al.; (1992); Proportional Mortality Ratio ("PMR") study	3084 white male FF deaths	1940-88 (49 yrs.)	U.S.A. white males	Chicago, Illinois, U.S.A.	not applicable	not applicable	not stated	Analysis done for IDSP: Kidney: between 1948 and 1982: PMR 1.648; (1.058-2.452); no dose-response analyses
L'ABBÉ & TOMLINSON (1992); (IDSP study); standardized mortality ratio ("SMR") study	5,414 males; 5,373 for duration of employment analysis	1950-89 (40 yrs.)	Ontario males	Metropolitan Toronto	strong for FFs under 40 and for FFs with less than 10 years since first exposure	SMR 94; (88-101); n=777	Kidney and ureter: SMR 43; (5-156); n=2; no dose-response is shown	
DEMERS et al.; (1992); standardized mortality ratio ("SMR") study	4546 males	1945-89 (45 yrs.)	1) U.S. white males; 2) 3676 police from Seattle & Tacoma, Wash., U.S.A. (white males)	Seattle & Tacoma, Wash, U.S.A.	yes, except for diseases of the arteries, veins & pulmonary circulation in FFs with 30 or more years of exposure	SMR 0.81; (0.77-0.86); n=1169	Kidney: SMR 27; (0.03-0.97); n=2; no dose-response analysis for this cause	com-pares FFs with police, who are also screened for physical health
GUIDOTTI; (1993); standardized mortality ratio ("SMR") study	3328	1927-87 (61 yrs.)	Alberta males	Edmonton & Calgary, Alberta	no, probably because: 1-Alberta males are unusually healthy; and, 2-most subjects were traced	SMR 96.2; (86.6-106.5); n=370	Kidney and ureter: SMR 414 ($p<0.05$); (166.4-853.1); n=7; possible dose-response	
BEAUMONT et al.; (1991); mortality rate ratio ("RR") study	3066 white males	1940-82 (43 yrs.)	U.S.A. white males	San Francisco, Calif., U.S.A.	yes	RR 0.90; (0.85-0.95); n=1186	Kidney: RR 0.68 (0.19-1.74); n=4; no dose-response analysis for this cause	
VENA & FIEDLER; (1987); standardized mortality ratio ("SMR") study	1867 white males	1950-79 (30 yrs.)	U.S.A. white males	Buffalo, N.Y., U.S.A.	yes, especially for circulatory system diseases.	SMR 0.95; (0.87-1.04); n=470	Kidney: SMR 1.30; (0.26-3.80); n=3; no dose-response analysis for this cause	
TORNLING et al.; (1994); standardized mortality ratio ("SMR") study	1116	1951-86 (36 yrs.)	Stockholm males	Stockholm, Sweden	yes	SMR 82; (73-91); n=316	Kidney: SMR 110; (30-281); n=4; possible dose-response	

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	KIDNEY CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COMMENTS
MASTRO-MATTEO; (1959); standardized mortality ratio ("SMR") study	1039	1921-53 (33 yrs.)	Toronto & Ontario males	Toronto, Ontario	not mentioned	significant excess of deaths for all causes compared to Toronto males	no significant increase or decrease in cancer deaths; no dose-response analysis	
MILHAM; (1983); proportional mortality ratio ("PMR") study	not stated	1950-79 (30 yrs.)	Washington state males	state of Washington, U.S.A.		not applicable	Kidney: PMR 115; (50-227); n=8; no dose-response analysis	PMR for 1950-79: 215; (79-468); n=6
MUSK et al.; (1978); standardized mortality ratio ("SMR") study	5655 males	1915-75 (61 yrs.)	male population of Mass. and U.S.A.	Boston, Mass., U.S.A.	yes	2470 deaths; 91% of expected for Mass. males; 94% of expected for U.S.A. white males	"Genitourinary": SMR 92; (71-117); n=64; no dose-response analysis	Genitourinary includes kidney, urinary and bladder cancers
ELIOPULOS et al.; (1984); standardized mortality ratio ("SMR") and proportionate mortality ratio ("PMR") study	990	1939-78 (about 40 yrs.)	Western Austr. males	Western Australia	yes	SMR 0.80; (0.67-0.96); n=116	"Genitourinary": PMR: 1.08; (0.29 - 2.76); n=4; (SMR not given); no dose-response analysis	types of cancer included in the PMR for genitourinary cancers are not specified
GRIMES et al.; (1991); proportional mortality ratio ("PMR") study	205 males	1969-88 (20 yrs.)	20 year and older Hawaiian males	Honolulu, Hawaii, U.S.A.	mentioned by authors	not applicable	"Genito-urinary system": Risk Ratio 2.28; (1.28-4.06); no dose-response analysis	"genito-urinary" includes bladder, kidney, ureter, prostate, testis and others

Fig. 26


Rectal Cancer Findings
L'Abbe (171 SMR, n=13) rectum/rectosigmoid junction
Demers (95 SMR, n=8) comp. to US males
Guidotti (161.4 SMR, n=14)
Heyer (65 SMR, n=2)
Vena&Fiedler (208 SMR, n=7)
Lewis (58.9 SMR, n=4) over 70
Lewis (29.5 SMR, n=3) under 70
Tornling (207 SMR, n=8)
Orris (164 PMR)
Decoufle (230 PMR, n=17)
Milham (103 PMR, n=11)
Sama (135 OR, n=135) comp. to state
Sama (97 OR) comp. to police
Beaumont (145 RR, n=13)
Demers (89 IDR) comp. to police
0
50
100
150
200
250
Legend
Standardized Mortality Ratio (SMR)
Proportional Mortality Ratio (PMR)
Odds Ratio (OR)
Relative Risk Ratio (RR)
Incidence Density Ratio (IDR)

(viii) Rectal cancer

It is estimated that in 1993 colorectal cancer accounted for 14% of new cancer cases and 10.1% of cancer deaths among adult males in Canada [130].

Mortality and morbidity studies

Two studies found a statistically significant increase in deaths from cancer of the rectum among firefighters [147, 31]. Seven studies found elevations, but none of those increases reached statistical significance [86, 202, 121, 59, 10, 195, 169]. Three studies found non-significant increases or decreases [32, 68, 109]. One found a statistically significant decrease in firefighters under 70 years of age [98]. Four others did not report results for this cause of death [128, 43, 63, 47]. None of the four studies which conducted dose-response analyses identified such a trend.

The findings of thirteen studies of rectal cancer in firefighters are illustrated in Fig. 26. More detailed is given in Table 17, which appears at the end of this section.

Statistically significant increases

The largest firefighter study, by Orris et al. (1992), examined the causes of 3084 firefighter deaths and found a PMR of 164 (95% CI 114-230) for rectal cancer mortality.

Another mortality study, by Decoufle et al. (1977), which reported twice the expected rate of death from this cause, combined firefighters with stationary engineers and hence it provides less than ideal data about firefighters.

Statistically significant decrease

Lewis and co-workers (1982) found a statistically significant decrease in rectal cancer mortality among Los Angeles firefighters under age 70, reporting an SMR of 29.5 (95% CI 6-86; n=3). The rate for those over age 70 remained decreased but not to a statistically significant degree (SMR 58.9; 95% CI 16-151; n=4).

Other findings

Non-statistically significant increases were found in six other studies.

Four studies found no increase or found decreases which did not reach statistical significance.

Dose-response analyses

No dose-response trend appeared in the findings in the IDSP cohort of 5414 Toronto firefighters for rectal cancer [86].

Tornling et al. (1994) noted SMRs of 275 in men with fewer than 20 years of employment and 293 in those with over 30 years; however, the SMRs *decreased* with the number of fires attended.

Nor was such a trend found in the other two studies which provided dose-response analyses for rectal cancer, conducted by Guidotti (1993) and Beaumont et al. (1991).

Potential causative agents

Dr. McDiarmid reported that an excess of rectal cancer

> " ... may easily be explained by the well documented exposure of firefighters to asbestos. Asbestos is well documented as an occupational carcinogen causing excesses of gastro intestinal cancers as a whole and for cancer of the stomach, colon and rectum in particular (Miller, 1978)." [111]

As mentioned previously, IARC has classified asbestos as a Group 1 carcinogen noting studies which found an approximately two-fold risk of rectal cancer among chrysotile asbestos production workers [79].

None of the studies reviewed included asbestos in their analyses of airborne contaminants at fire sites. Since asbestos has been widely used in the manufacture of various building insulation materials, it is anticipated that firefighters may be significantly exposed to airborne asbestos fibres at fires where such materials are present. Markowitz et al. (1980) reported evidence that firefighters are at risk for scarring of the lungs and pleura due to occupational asbestos exposure.

Summary of the Evidence

- While two studies found statistically significant increases in rectal cancer mortality, one of them combined firefighters with stationary engineers making it impossible to know the true extent of mortality from this cause among the firefighters; the other reported a PMR of 164.

- Increases were not consistently found among studies and one found a statistically significant decrease.

- No increase with duration of employment was identified in the four studies which provided dose-response analyses. As mentioned previously, dose-response analyses using duration of employment as a surrogate for exposure have too little information about the dose to allow an informed judgment about a response. Studies which conduct analyses based upon actual exposure and sufficient numbers of cases inspire somewhat more confidence. One study conducted an analysis based upon number of fires attended and did not find a dose-response trend. It, however, involved a small number of cases and, accordingly, must be regarded as uninformative.

- Asbestos exposure may occur in the course of firefighters' work and there is evidence that asbestos is a rectal carcinogen.

The Panel's conclusions and finding

Applying the Bradford Hill criteria, the weight of evidence does not support a probable connection between rectal cancer and working as a firefighter, although an occupational association is biologically plausible for firefighters exposed to asbestos. While there is a statistically significant increase in one study, this was not consistently found among studies. The available dose-response analyses are uninformative.

In summary, a single study that shows a statistically significant increase in this cancer among firefighters is not sufficient evidence to establish a probable connection between rectal cancer and the occupation of firefighting. Accordingly, the Panel makes the following finding.

- **There is not a probable connection between rectal cancer and the occupation of firefighting.**

The Panel's recommendation

There may be circumstances in which a firefighter contracts rectal cancer due to work, hence the Panel makes the following recommendation.

- **The Panel recommends that the WCB continue to adjudicate claims for rectal cancer from firefighters having regard for the individual merits of each claim.**

TABLE 17: FIREFIGHTER MORTALITY/MORBIDITY STUDIES: RECTAL CANCER FINDINGS

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	RECTAL CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
ORRIS, et al.; (1992); proportional mortality ratio ("PMR") study	3084 white male FF deaths	1940-88 (49 yrs.)	U.S.A. white males	Chicago, Illinois, U.S.A.	Not applicable	Not applicable	Rectum: PMR 164; (114-230); n=not given; no dose-response analyses	
L'ABBÉ & TOMLINSON (1992); (IDSP study); standardized mortality ratio ("SMR") study	5,414 males; 5,373 for duration of employment analysis	1950-89 (40 yrs.)	Ontario males	Metropolitan Toronto	strong for FFs under 40 and for FFs with less than 10 years since first exposure	SMR 94; (88-101); n=777	Rectum and rectosigmoid junction: SMR 171; (91-293); n=13; dose-response not apparent	
DEMERS et al.; (1992); standardized mortality ratio ("SMR") study	4546 males	1945-89 (45 yrs.)	1) U.S. white males; 2) 3676 police from Seattle & Tacoma, Wash., U.S.A. (white males)	Seattle & Tacoma, Wash, U.S.A.	yes, except for diseases of the arteries, veins & pulmonary circulation in FFs with 30 or more years of exposure	SMR 0.81; (0.77-0.86); n=1169	Rectum: SMR compared to US males: 0.95; (0.41-1.87); n=8; Incidence Density Ratio compared to police: 0.89; (0.30-2.66); no dose-response analysis for this cause	compares FFs with police, who are also screened for physical health
GUIDOTTI; (1993); standardized mortality ratio ("SMR") study	3328	1927-87 (61 yrs.)	Alberta males	Edmonton & Calgary, Alberta	no, probably because: 1-Alberta males are unusually healthy; and, 2-most subjects were traced	SMR 96.2; (86.6-106.5); n=370	Colon and rectum: SMR 161.4; (88.3-270.9); n=14; dose-response not apparent	
BEAUMONT et al.; (1991); mortality rate ratio ("RR") study	3066 white males	1940-82 (43 yrs.)	U.S.A. white males	San Francisco, Calif., U.S.A.	yes	RR 0.90; (0.85-0.95); n=1186	Rectum:: RR 1.45; (0.77-2.49); n=13; dose-response not shown	

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	RECTAL CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COMMENTS
HEYER et al.; (1990); standardized mortality ratio ("SMR") study	2289 males	1945-83 (39 yrs.)	U.S.A. white males	Seattle, Wash., U.S.A.	yes; survivor effect also noted.	SMR 76; (69-85); n=383	Rectum: SMR 65; (8-237); n=2; no dose-response analysis for this cause	
VENA & FIEDLER; (1987); standardized mortality ratio ("SMR") study	1867 white males	1950-79 (30 yrs.)	U.S.A. white males	Buffalo, N.Y., U.S.A.	yes, especially for circulatory system diseases.	SMR 0.95; (0.87-1.04); n=470	Rectum: SMR 2.08; (0.83-4.28); n=7; no dose-response analysis for this cause	
LEWIS et al.; (1982); standardized cancer mortality ratio ("SMR") study	1559	1940-80 (41 yrs.)	U.S.A. white males	Los Angeles, Calif.	yes	declining	Rectum: SMR for under age 70: 29.5; (6-86); n=3; 70 and over: 58.9; (16-151); n=4; no dose-response analysis	mention of any cancer on death certificate was coded as a cancer death; this may overestimate cancer rate
TORNLING et al.; (1994); standardized mortality ratio ("SMR") study	1116	1951-86 (36 yrs.)	Stockholm males	Stockholm, Sweden	yes	SMR 82; (73-91); n=316	Rectum: SMR 207; (89-406); n=8; no dose-response shown	
DECOUFLE et al., (1977); proportionate mortality ("PMR") study	1113 stationary engineers and firefighters	1967 (1 year)	U.S. PMRs	4 areas of U.S.	not applicable	not applicable	Rectum: PMR 2.30; (1.34-3.68); ($p<0.01$); n=17; no dose-response analysis	combines findings for engineers and firefighters which may dilute or overestimate results
MASTRO-MATTEO; (1959); standardized mortality ratio ("SMR") study	1039	1921-53 (33 yrs.)	Toronto & Ontario males	Toronto, Ontario	not mentioned	significant excess of deaths for all causes compared to Toronto males	no significant increase or decrease in cancer deaths; no dose-response analysis	
SAMA et al.; (1990); case-control study	321 FF cancer cases	1982-86 (5 yrs.)	1) 29,277 Massachusetts males; 2) 392 police	state of Massachusetts, U.S.A.	not applicable	not applicable	Rectum: Odds Ratio compared to police: 97; (50-188); compared to state: 135; (84-219); n=22; no dose-response analysis	compares FFs with general population and with police, who are also screened for physical health

AUTHOR(S); YEAR; TYPE OF STUDY	NUMBER OF FIRE FIGHTERS	YEARS STUDIED	COMPARISON CONTROL GROUP	GEOGRAPHICAL LOCATION	HEALTHY WORKER EFFECT IDENTIFIED?	OVERALL MORTALITY - S.M.R. (95% C.I.)	RECTAL CANCER - S.M.R.; (95% C.I.); number of cases; dose-response?	COM-MENTS
MILHAM; (1983); proportional mortality ratio ("PMR") study	not stated	1950-79 (30 yrs.)	Washington state males	state of Washington, U.S.A.	not applicable	not applicable	Rectum: PMR 103; (51-184); n=11; no dose-response analysis	PMR for 1970-79: 194; (71-422); n=6

CHAPTER 4. SUMMARY OF THE PANEL'S FINDINGS AND RECOMMENDATIONS

Regarding cardiovascular disease:

- A probable connection exists between cardiovascular disease and the occupation of firefighting.

- When the Board adjudicates a claim for heart disease from a firefighter, occupation should be recognized as a risk factor for cardiovascular disease and should be weighed with the other risk factors such as hypertension, smoking and family history when determining entitlement under the *Workers' Compensation Act*.

Regarding aortic aneurysm:

- A probable connection exists between firefighting and atherosclerosis which results in aortic aneurysm.

- "Atherosclerosis which results in aortic aneurysm" and the associated process, trade or occupation of "firefighter" should be added to Schedule 3 of the *Act*.

- A rebuttal matrix approved by the Panel should be used to assess the evidence used to rebut the presumption.

Regarding lung cancer:

- There is not a probable connection between lung cancer and the occupation of firefighting.

- The Panel recommends that the WCB continue to adjudicate claims for lung cancer from firefighters having regard for the individual merits of each claim.

Regarding brain cancer:

- A probable connection exists between firefighting and primary cancer of the brain.

- "Primary cancer of the brain" and the associated process, trade or occupation of "firefighter" should be added to Schedule 3 of the Act.

- A rebuttal matrix approved by the Panel should be used to assess the evidence used to rebut the presumption.

Regarding lymphatic and haematopoietic cancers:

- A probable connection exists between firefighting and primary lymphatic and haematopoietic cancers.

- "Primary lymphatic or haematopoietic cancer" and the associated process, trade or occupation of "firefighter" should be added to Schedule 3 of the Act.

- A rebuttal matrix approved by the Panel should be used to assess the evidence used to rebut the presumption.

Regarding colon cancer:

- A probable connection exists between colon cancer and the occupation of firefighting.

- Because a probable connection has been established, guidelines developed and approved by the Panel should be used to assist adjudicators in assessing the merits of each claim for colon cancer from firefighters.

Regarding bladder cancer:

- A probable connection exists between primary bladder cancer and the occupation of firefighting.

- Because a probable connection has been established, guidelines developed and approved by the Panel should be used to assist adjudicators in assessing the merits of claims for bladder cancer from firefighters.

Regarding kidney cancer:

- A probable connection exists between kidney cancer and the occupation of firefighting.

- Because a probable connection has been established, guidelines developed and approved by the Panel should be used to assist adjudicators in assessing the merits of claims for kidney cancer from firefighters.

Regarding rectal cancer:

- There is not a probable connection between rectal cancer and the occupation of firefighting.

- The Panel recommends that the WCB continue to adjudicate claims for rectal cancer from firefighters having regard for the individual merits of each claim.

Further Panel investigations:

Occupational non-malignant respiratory disease and hearing loss among firefighters will be addressed in subsequent Panel Reports.

APPENDIX A:

IARC EVALUATION OF EVIDENCE FOR CARCINOGENICITY

(a) Degrees of evidence for carcinogenicity to humans and to experimental animals and supporting evidence

It should be noted that these categories refer only to the strength of the evidence that these agents are carcinogenic and not to the extent of their carcinogenic activity (potency) nor to the mechanism involved. The classification of some agents may change as new information becomes available.

(i) Human carcinogenicity data

The evidence relevant to carcinogenicity from studies in humans is classified into one of the following categories:

Sufficient evidence of carcinogenicity: The Working Group considers that a causal relationship has been established between exposure to the agent and human cancer. That is, a positive relationship has been observed between exposure to the agent and cancer in studies in which chance, bias and confounding could be ruled out with reasonable confidence.

Limited evidence of carcinogenicity: A positive association has been observed between exposure to the agent and cancer for which a causal interpretation is considered by the Working Group to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence.

Inadequate evidence of carcinogenicity: The available studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding the presence or absence of a causal association.

Evidence suggesting lack of carcinogenicity: There are several adequate studies covering the full range of doses to which human beings are known to be exposed, which are mutually consistent in not showing a positive association between exposure to the agent and any studied cancer at any observed level of exposure. A conclusion of "evidence suggesting lack of carcinogenicity" is inevitably limited to the cancer sites, circumstances and doses of exposure and length of observation covered by the available studies. In addition, the possibility of a very small risk at the levels of exposure studied can never be excluded.

In some instances, the above categories may be used to classify the degree of evidence for the carcinogenicity of the agent for specific organs or tissues.

(ii) Experimental carcinogenicity data

The evidence relevant to carcinogenicity in experimental animals is classified into one of the following categories:

Sufficient evidence of carcinogenicity: The Working Group considers that a causal relationship has been established between the agent and an increased incidence of malignant neoplasms or of an appropriate combination of benign and malignant neoplasms (as described on p.23) in (a) two or more species of animals or (b) in two or more independent studies in one species carried out at different times or in different laboratories or under different protocols.

Exceptionally, a single study in one species might be considered to provide sufficient evidence of carcinogenicity when malignant neoplasms occur to an unusual degree with regard to incidence, site, type of tumour or age at onset.

In the absence of adequate data on humans, it is biologically plausible and prudent to regard agents for which there is sufficient evidence of carcinogenicity in experimental animals as if they presented a carcinogenic risk to humans.

Limited evidence of carcinogenicity: The data suggest a carcinogenic effect but are limited for making a definitive evaluation because, e.g., (a) the evidence of carcinogenicity is restricted to a single experiment; or (b) there are unresolved questions regarding the adequacy of the design, conduct or interpretation of the study; or (c) the agent increases the incidence only of benign neoplasms or lesions of uncertain neoplastic potential, or of certain neoplasms which may occur spontaneously in high incidences in certain strains.

Inadequate evidence of carcinogenicity: The studies cannot be interpreted as showing either the presence or absence of a carcinogenic effect because of major qualitative or quantitative limitations.

Evidence suggesting lack of carcinogenicity: Adequate studies involving at least two species are available which show that, within the limits of the tests used, the agent is not carcinogenic. A conclusion of evidence suggesting lack of carcinogenicity is inevitably limited to the species, tumour sites and doses of exposure studied.

(iii) Supporting evidence of carcinogenicity

The other relevant data judged to be of sufficient importance as to affect the making of the overall evaluation are indicated.

(b) Overall evaluation

Finally, the total body of evidence is taken into account; the agent is described according to the wording of one of the following categories, and the designated group is given. The categorization of an agent is a matter of scientific judgement, reflecting the strength of the evidence derived from studies in humans and in experimental animals and from other relevant data.

Group 1 - The agent is carcinogenic to humans.

The category is used only when there is sufficient evidence of carcinogenicity in humans.

Group 2

This category includes agents for which, at one extreme, the degree of carcinogenicity in humans is almost sufficient, as well as agents for which, at the other extreme, there are no human data but for which there is experimental evidence of carcinogenicity. Agents are assigned to either 2A (probably carcinogenic) or 2B (possibly carcinogenic) on the basis of epidemiological, experimental and other relevant data.

Group 2A - The agent is probably carcinogenic to humans.

This category is used when there is limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals. Exceptionally, an agent may be classified into this category solely on the basis of limited evidence of carcinogenicity in humans or of sufficient evidence of carcinogenicity in experimental animals strengthened by supporting evidence from other relevant data.

Group 2B - The agent is possibly carcinogenic to humans.

This category is generally used for agents for which there is limited evidence in humans in the absence of sufficient evidence in experimental animals. It may also be used when there is inadequate evidence of carcinogenicity in humans or when human data are nonexistent but there is sufficient evidence of carcinogenicity in experimental animals. In some instances, an agent for which there is inadequate evidence or no data in humans but limited evidence

of carcinogenicity in experimental animals together with supporting evidence from other relevant data may be placed in this group.

Group 3 - The agent is not classifiable as to its carcinogenicity to humans.

Agents are placed in this category when they do not fall into any other group.

Group 4 - The agent is probably not carcinogenic to humans.

This category is used for agents for which there is evidence suggesting lack of carcinogenicity in humans together with evidence suggesting lack of carcinogenicity in experimental animals. In some circumstances, agents for which there is inadequate evidence of or no data on carcinogenicity in humans but evidence suggesting lack of carcinogenicity in experimental animals, consistently and strongly supported by a broad range of other relevant data, may be classified in this group.

APPENDIX B: GLOSSARY

Ascertainment bias:

The results of epidemiological studies can sometimes be skewed by an *ascertainment bias*. This may occur when the total cohort is not (or usually, *cannot* be) traced in order to identify illness or death; in other words, they are "lost to follow-up". In order to err on the side of caution, epidemiological studies assume that subjects lost to follow-up are alive. This means that they contribute "person-years" throughout the study period even if they have died. This has the effect of overestimating the time subjects worked and remained healthy as well as underestimating the "observed" number of deaths. If the outcomes for those lost to follow-up *were* known, their deaths would raise the mortality rates in one or more categories of disease, because death is a common reason for the inability to trace members of a cohort.

Whenever fewer than 100% of the cohort is traced, an ascertainment bias will cause an underestimation of risk, unless the author specifically adjusts for this bias. The larger the number of subjects who are not traced, the larger the underestimation of risk.

CEV (Ceiling Exposure Value):

The maximum airborne concentration of a chemical to which a worker may be exposed at any time during a workday.

carboxyhemoglobin

Haemoglobin combined with carbon monoxide. Carbon monoxide occupies the sites on the haemoglobin molecule that normally bind with oxygen and is not readily displaced from the molecule.

Case-control study:

A "case-control study" in the context of epidemiology is a study design which compares the exposure of cases (death or disease) with the exposure of age- and sex-matched controls who do not have the disease or cause of death under investigation.

Cohort study:

A "cohort study" in the context of epidemiology is a study design which traces the mortality or morbidity of a group of people having in common an exposure to a potentially hazardous substance, eg. an occupational group.

Dose-response:

A dose-response trend is shown when an increase in the "dose" (exposure level, intensity or duration) corresponds to an increase in the "response" (death or disease).

Epidemiology:

The study of disease patterns in groups of people. In the context of IDSP Reports, diseases are studied in order to determine if they reflect occupational exposures to potentially hazardous substances.

Grab sampling:

Short-term or instantaneous sample collection usually for the purposes of identifying the presence of specific contaminants in an environment and to define peak concentrations throughout an exposure period.

Healthy worker effect:

Most epidemiological studies compare workers with the general population because other more appropriate comparison groups are usually not available. Since the general population includes people who do not or cannot work due to illness or disability, a working population is usually healthier and is expected to have a lower mortality rate for most causes of death. The healthy worker effect may conceal a real increase in deaths among workers. Comparisons with another group of "healthy" workers, rather than with the general population, are therefore more likely to provide accurate estimates of occupational risks.

Whether or not the healthy worker effect influences cancer mortality ratios is controversial. In a previous Panel Report, the IDSP published comments on the healthy worker effect solicited from nine experts [75]. The Panel's review of those opinions led it to conclude that the healthy worker effect must be taken into account when interpreting epidemiological studies of mortality or morbidity from *any* cause, including cancer.

IAFF:

The International Association of Fire Fighters.

IARC:

The International Agency for Research on Cancer.

Incidence Density Ratio (IDR):

The ratio of the age-matched incidence of disease in one group to the age-matched incidence in the comparison group. For example, Demers et al. (1992) compared the incidence of aortic aneurysms among firefighters with the incidence of aortic aneurysms among police in the same age groups.

Knockdown phase of firefighting:

The process of extinguishing the main fire.

Latency period:

The period of time between exposure to a substance(s) and the appearance of the disease which it has caused.

Null hypothesis:

The null hypothesis states that the results observed in a study, experiment or test are no different from what might have occurred as a result of chance alone.

OPFFA:

The Ontario Professional Fire Fighters Association.

Odds Ratio (OR):

"Case-control" studies, as opposed to cohort studies, report their results in terms of an odds ratio (OR). This represents the likelihood that observed cases had a certain exposure, compared to the likelihood that controls had that exposure. An equal likelihood is expressed as 1.

Overhaul phase of firefighting:

Searching for and extinguishing hidden fires after the main fire has been extinguished.

PFOFF:

The Provincial Federation of Ontario Fire Fighters.

p-value (eg. "p<0.05"):

This refers to the probability that chance produced an apparent excess of disease. For example, "p<0.05" means there is less than a 5% likelihood that the observed result could have occurred by chance.

Problem of multiple tests of significance:

When numerous disease outcomes are examined, positive associations will occur for some of them by chance alone. Caution must be exercised in interpreting the results, especially if there was no prior hypothesis that a particular disease would be elevated in an occupational group.

Proportionate Mortality Ratio (PMR):

These are used in studies which do not have a count of the exposed population (denominator) to allow the calculation of SMRs. The PMR is the ratio of the proportion of observed deaths among firefighters due to a disease compared with the proportion of deaths due to that disease in an age-matched comparison group. A PMR of more than 100 suggests an excess risk, similar to an SMR, as discussed above.

SCBA (Self-Contained Breathing Apparatus):

A pressurized bottle of air carried on the firefighter's back. A hose leading from the air tank feeds clean air into a mask covering the face.

STEV (Short-Term Exposure Value):

A 15-minute time-weighted average exposure concentration which may not be exceeded at any time during a workday.

Standardized Mortality Ratio (SMR):

This Report includes a discussion of epidemiological studies which identify causes of death that occurred more often for firefighters than for the groups to which they were compared. Results are measured in terms of a "standardized mortality ratio". An SMR is an estimate computed by comparing the number of deaths **observed** (i.e., that *occurred*) among firefighters with the number of deaths which are **expected** based upon a comparison group of the same age and sex, during the same time period:

$$\textbf{SMR} = \frac{\text{"observed" deaths among firefighters}}{\text{"expected" deaths}} \times 100$$

Most authors multiply the ratio by 100, but some do not. An SMR greater than 100 (or 1) suggests an excess risk of death. Epidemiologists evaluate the ***statistical significance*** of an elevated SMR by using the *95% confidence interval*, which is the range in which the true SMR would fall 95% of the time.

If the lower end of the 95% confidence interval is **above** 100 (or 1), the likelihood that the excess mortality is due to chance is less than 5% (or 1 out of 20). If the SMR is **under** 100 (or 1) and the upper 95% end of the confidence interval is under 100 (or 1), a statistically significant **decrease** in deaths is indicated, compared to the number of deaths that would normally be expected.

Survivor effect:

Because adequate health is required for workers to continue working, and unwell workers may leave work before reaching retirement, it is possible that only the healthiest workers continue to be employed for a long term. If duration of employment is the only available measure of exposure, and if there is a survivor effect, a dose-response may be masked.

TWAEV (Time-Weighted Average Exposure Value):

The average airborne concentration of a chemical agent to which a worker may be exposed in a workday or work week.

APPENDIX C:
WCB OPERATIONAL POLICY - Heart Conditions

Policy

The WCB accepts claims as work related when:

- a causal relationship is shown between the cardiac condition and an accident at work, or
- the cardiac condition is established as a disablement "arising out of and in the course of employment."

Guidelines

The Board accepts entitlement for cardiac conditions under any of the following circumstances:

- traumatic injury, either penetrating or non-penetrating injuries to the chest wall,
- electric shock producing irregular cardiac rhythm,
- inhalation of smoke and various noxious gases and fumes, e.g., fire fighters
- complication of treatment for a work-related injury, e.g., anaesthesia with an interval of hypotension, hypoxia or cardiac arrest

note: When entitlement is established under the above points for a cardiac condition, there will be no limitation of ongoing entitlement as long as the subsequent condition is related to the work-related cardiac condition,

or

- unusual physical exertion for the individual and/or acute emotional stress with no significant delay in the onset of symptoms.

note: This instance is allowed on the basis of aggravation of a pre-existing non-work-related condition. When entitlement is established, the condition has stabilized, and a Permanent Disability evaluation has been conducted, further entitlement will not be granted for a subsequent cardiac condition unless there is a new work-related occurrence, which merits allowance under a new claim.

Temporary Disability

In most instances, claims for cardiac conditions are considered on the basis of aggravation of a pre-existing condition, usually arteriosclerotic heart disease.

When a claim is accepted under this category for a heart condition, entitlement includes full benefits for compensation and health care.

Permanent Disability

Permanent disability awards are granted in cardiac claims when permanent disability results from the injury.

The Claims Adjudicator obtains the Occupational Medicine Consultant's opinion on the suitability of permanent disability evaluation, based on the work-related condition. The Consultant may then arrange an assessment, including stress testing, for details on the worker's limitations, with an outside Cardiologist.

When the test results are received, a further opinion is obtained from the Occupational Medicine Consultant. If the condition is then considered suitable for permanent disability assessment, an examination is conducted by the Board's Permanent Disability (Pensions) Medical Consultant to evaluate the degree of clinical impairment. The American Medical Association Classification of Cardiovascular Impairment may be used as a guide.

The Pensions Adjudicator makes a determination using the assessment results, interviews the worker, processes the award where applicable, and confirms the details by letter.

Indisputable Rating

Where the worker is 100% permanently totally disabled and unfit for evaluation, an "Indisputable Rating" may be required based on review of all medical information.

Traumatic Cardiac Conditions

In the case of traumatic cardiac conditions, e.g.

- cardiac tamponade, defined as compression of the heart by pericardial fluid,
- cardiac contusion
- valvular damage, or
- effects of the passage of an electrical current,

the appropriate award for Permanent Disability, determined by the Pensions Adjudicator, is granted to the worker. There is no limitation of ongoing entitlement in the claim, relative to the compensable cardiac condition.

Aggravation of Pre-existing Conditions

Certain limitations apply when determining the Permanent Disability award in claims where allowance has been made on the basis of aggravation of a pre-existing non-work-related condition (see Second Injury and Enhancement Fund, 08-01-05).

Where a pre-existing disability is considered "major" or "moderate", the amount of the permanent disability award may be reduced.

Subsequent to Permanent Disability assessment, ongoing medical treatment is not within entitlement if the need for treatment arises from an underlying vascular disease which pre-existed the accident. Exceptions are those cases where various surgical procedures, e.g. coronary artery bypass, may be implemented in the acute phase of the disability. Ongoing entitlement may be extended on the advice of the Board's Occupational Medicine Consultant.

Fatal Claims

If death occurs immediately, e.g.,

- as the result of the compensable cardiac condition, shortly after the initial onset, or
- while the condition is still in the acute phase,

the fatal claim may be accepted and full death benefits may be paid.

If death occurs as the result of a new or progressive cardiac condition, the fatal claim is not accepted unless there is a new occurrence, at work, which merits allowance under a new claim.

Claims Not Meeting General Criteria

Cardiac claims which do not meet the general criteria shall be individually judged on their own merits. The benefit of doubt applies.

REFERENCES

Legislative Authority	Workers' Compensation Act Sections 1(1)(a), 36, 45
Minute	Board of Directors, #13, April 8, 1980, page 4845 #22, November 25, 1980, page 4878

APPENDIX D:

The following questions were asked of Dr. James Melius, Dr. C. D. Morgan and Dr. J. K. Wilson in letters dated May 4, 1992:

1. Please explain what an aortic aneurysm is.

2. The study found a consistently high rate of aortic aneurysms among these firefighters. Can you provide an explanation of this finding?

3. Excess deaths from aortic aneurysms were found in all age groups. The excess was more significant in those over 60 years of age. Risk was reportedly higher only for terminated workers. In your opinion, is there a biological process which is rooted in their occupational exposures and/or stressors which could have set the stage for aortic aneurysms to occur after they has stopped working as firefighters?

4. While all the potential exposures these firefighters could have encountered are not known, the Guidotti and Clough review identified several common exposures. In your opinion, could these exposures cause or potentiate aortic aneurysms or other cardiac conditions and if so, how?

5. In your opinion, what biological reasons could account for the excess rates of arteriosclerosis among these firefighters?

6. We know that cigarette smoke has a significant impact on the vascular system. Would high intensity periods of smoke inhalation of short duration have a similar adverse effect on the vascular system?

7. We understand that firefighters may be relatively sedentary for periods of time, but that they are then called into sudden vigourous activity when an alarm occurs. Please comment on any cardiovascular effects which this could cause in the short or long term.

8. Please comment on the effect that shiftwork could have on cardiovascular health.

9. It appears that a "healthy worker effect" is present early in the careers of firefighters but that later in their careers, this effect is eliminated. Would you please provide your comments on this apparent trend.

10. Some US jurisdictions presume that certain cardiovascular diseases in firefighters are related to their occupational exposures. Given patterns in epidemiologic studies and your expertise, can you offer any information which would support or refute that connection?

11. Although our study did not show a significant increase in the incidence of myocardial infarction, firefighters have long regarded it as an important concern. Would you comment on any ways in which their occupational environment might affect the incidence of myocardial infarction.

12. Do you think that mortality studies provide accurate indicators of all health problems which are caused or exacerbated by occupation? Why or why not?

Since the Panel is anxious to have a comprehensive understanding of these issues, any other comments which you might like to make about the risk of cardiovascular disease and the hazards of firefighting will be appreciated.

APPENDIX E:

The following questions were asked of Dr. M. M. McDiarmid, Dr. N. F. Boyd and Dr. I. Quirt in letters dated May 4, 1992:

1. This study found a significant increase in risk of cancer of the brain and other nervous system tumours. Please identify any process related to firefighting which could account for this increase.

2. Are latency periods generally associated with the development of brain and other nervous system cancers between 0 to 19 years since first exposure and between 40 to 59 years since first exposure?

3. Would you also comment on the finding of increased brain and other nervous system cancers in firefighters with less than 15 and more than 30 years of employment.

4. While all the potential exposures these firefighters could have encountered are not known, the Guidotti and Clough review identified several common exposures. In your opinion, could these exposures cause or potentiate any of the cancers whose rates were elevated, such as cancer of the testis (SMR 246); lymphatic leukaemia (SMR 190); cancer of the rectum and rectosigmoid junction (SMR 171); lymphosarcoma and reticulum cell sarcoma (SMR 150); cancer of the pancreas (SMR 140), the pharynx (SMR 139) and the prostate (SMR 132). If so, how?

5. Firefighters have long been concerned about an increase in their risk of lung cancer. Our study did not find such an increase (SMR 95). Do you have any information or comments about this?

6. It appears that a "healthy worker effect" is present early in the careers of firefighters but that later in their careers, this effect is eliminated. Would you please provide your comments on this apparent trend.

7. Some U.S. jurisdictions presume that certain types of cancer in firefighters are related to their occupational exposures. Given patterns in epidemiologic studies and your knowledge of carcinogenesis, can you offer any information which would support or refute that connection?

8. Do you think that mortality studies provide accurate indicators of all health problems which are caused or exacerbated by occupation? Given the exposures identified in the literature, are there some types of cancer which would be better studied in a morbidity study?

Since the Panel is anxious to have a comprehensive understanding of these issues, any other comments which you might like to make about the risk of cancer and the hazards of firefighting will be appreciated.

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